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L111 ANSWER 1 OF 34     MEDLINE
AN   97211013     MEDLINE
DN   97211013   PubMed ID: 9058011
TI   p-Hydroxybenzyl alcohol attenuates learning deficits in the inhibitory
     avoidance task: involvement of serotonergic and dopaminergic systems.
AU   Wu C R; Hsieh M T; Liao J
CS   Institute of Chinese Pharmaceutical Sciences, China Medical College,
     Taichung, Taiwan, ROC.
SO   CHINESE JOURNAL OF PHYSIOLOGY, (1996) 39 (4) 265-73.
     Journal code: 7804502. ISSN: 0304-4920.
CY   TAIWAN: Taiwan, Province of China
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   199705
ED   Entered STN: 19970609
     Last Updated on STN: 19970609
     Entered Medline: 19970523
AB   p-Hydroxybenzyl alcohol (HBA), an aglycone of gastrodin, is an active
     ingredient of Gastrodia elata BLUME. In this study, we investigated the
     action of HBA on acquisition of an inhibitory avoidance response in rats
     and used piracetam as a positive control. The results indicated that
     scopolamine, a cholinergic receptor antagonist, injected before training
     impaired retention. HBA did not attenuate the scopolamine-induced
     impairment, but piracetam did. p-Chloroamphetamine, a
     serotonin releaser, injected before training impaired retention.
     HBA at 5 mg/kg and piracetam at 100 mg/kg could counteract the p-
     chloroamphetamine-induced deficit. Apomorphine, a dopaminergic
     receptor agonist, also impaired retention. HBA at 5 mg/kg and
     piracetam at 300 mg/kg could ameliorate the apomorphine-induced
     amnesia. The above results indicated that HBA, different from
     piracetam, can attenuate impairments induced by p-
     chloroamphetamine and apomorphine, but had no effect on
     impairment induced by scopolamine in an inhibitory avoidance task
     in rats. Such findings suggest that HBA may act through suppressing
     dopaminergic and serotonergic activities and thus improves learning.
CT   Check Tags: Animal; Male; Support, Non-U.S. Gov't
        Apomorphine: PD, pharmacology
       *Avoidance Learning: PH, physiology
       *Benzyl Alcohols: TU, therapeutic use
       *Dopamine: PH, physiology
        Dopamine Agonists: PD, pharmacology
        Drug Combinations
        Electroshock
       *Learning Disorders: DT, drug therapy
        Motor Activity: DE, drug effects
        Muscarinic Antagonists: PD, pharmacology
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Rats
Rats, Sprague-Dawley
Reaction Time: DE, drug effects
Scopolamine: PD, pharmacology
*Serotonin: PH, physiology
Serotonin Agents: PD, pharmacology
p-Chloroamphetamine: PD, pharmacology
RN 50-67-9 (Serotonin); 51-34-3 (Scopolamine); 51-61-6 (Dopamine); 58-00-4 (Apomorphine); 623-05-2 (4-hydroxybenzyl alcohol); **64-12-0 (p-Chloroamphetamine)**
CN 0 (Benzyl Alcohols); 0 (Dopamine Agonists); 0 (Drug Combinations); 0 (Muscarinic Antagonists); 0 (Serotonin Agents)

L111 ANSWER 2 OF 34 MEDLINE
AN **96335663** MEDLINE
DN **96335663** PubMed ID: **8764668**
TI **Dextroamphetamine enhances** "neural network-specific" physiological signals: a positron-emission tomography rCBF study.
AU Mattay V S; Berman K F; Ostrem J L; Esposito G; Van Horn J D; Bigelow L B; Weinberger D R
CS Clinical Brain Disorders Branch, Intramural Research Porgram, National Institute of Mental Health, National Institutes of Health Neuroscience Center at Saint Elizabeth's, Washington, DC 20032, USA.
SO JOURNAL OF NEUROSCIENCE, **(1996 Aug 1)** 16 (15) 4816-22.
Journal code: 8102140. ISSN: 0270-6474.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Priority Journals
EM 199610
ED Entered STN: 19961106
Last Updated on STN: 19961106
Entered Medline: 19961022
AB Previous studies in animals and humans suggest that monoamines **enhance** behavior-evoked neural activity relative to nonspecific background activity (i.e., increase signal-to-noise ratio). We studied the effects of **dextroamphetamine**, an indirect monoaminergic agonist, on cognitively evoked neural activity in eight healthy subjects using positron-emission tomography and the O15 water intravenous bolus method to measure regional cerebral blood flow (rCBF). **Dextroamphetamine** (0.25 mg/kg) or placebo was administered in a double-blind, counterbalanced design 2 hr before the rCBF study in sessions separated by 1-2 weeks. rCBF was measured while subjects performed four different tasks: two abstract reasoning tasks--the Wisconsin Card Sorting Task (WCST), a neuropsychological test linked to a cortical network involving dorsolateral prefrontal cortex and other association cortices, and Ravens Progressive Matrices (RPM), a nonverbal intelligence test linked to posterior cortical systems--and two corresponding sensorimotor control tasks. There were no significant drug or task effects on pCO2 or on global blood flow. However, the effect of **dextroamphetamine** (i.e., **dextroamphetamine** vs placebo) on task-dependent rCBF activation (i.e., task - control task) showed double dissociations with respect to task and region in the very brain areas that most distinctly differentiate the tasks. In the superior portion of the left inferior frontal gyrus, **dextroamphetamine** increased rCBF during WCST but decreased it during RPM (ANOVA $F(1,7) = 16.72$, $p < 0.0046$). In right hippocampus, blood flow decreased during WCST but increased during RPM (ANOVA $F(1,7) = 18.7$, $p < 0.0035$). These findings illustrate that **dextroamphetamine** tends to "focus" neural activity, to highlight the neural network that is specific for a particular cognitive task. This capacity of **dextroamphetamine** to induce cognitively specific

signal augmentation may provide a neurobiological explanation for improved cognitive efficiency with **dextroamphetamine.**

CT Check Tags: Female; Human; Male
Adult
Analysis of Variance
*Brain: RI, radionuclide imaging
*Cerebrovascular Circulation: DE, drug effects
Cognition: DE, drug effects
***Dextroamphetamine: PD, pharmacology**
Memory: DE, drug effects
Tomography, Emission-Computed

RN **51-64-9 (Dextroamphetamine)**

L111 ANSWER 3 OF 34 MEDLINE
AN **96202295** MEDLINE
DN **96202295** PubMed ID: **8643648**
TI Adrenocortical suppression blocks the **memory-enhancing** effects of **amphetamine** and epinephrine.
AU Roozendaal B; Carmi O; McGaugh J L
CS Center for the Neurobiology of Learning and Memory, University of California, Irvine 92717-3800, USA.
NC MH12526 (NIMH)
MH14599 (NIMH)
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, **(1996 Feb 20)** 93 (4) 1429-33.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199607
ED Entered STN: 19960726
Last Updated on STN: 19960726
Entered Medline: 19960717
AB This study examined glucocorticoid-adrenergic interactions in modulating acquisition and **memory** storage for inhibitory avoidance training. Systemically (s.c.) administered **amphetamine** (1 mg/kg), but not epinephrine (0.1 mg/kg) or the peripherally acting **amphetamine** derivative 4-OH **amphetamine** (2 mg/kg), given to rats shortly before training facilitated acquisition performance in a continuous multiple-trial inhibitory avoidance (CMIA) task. Adrenocortical suppression with the 11beta-hydroxylase inhibitor metyrapone (50 mg/kg; s.c.), given to rats 90 min before training, did not block the effect of **amphetamine** and did not affect acquisition performance of otherwise untreated animals. Retention of CMIA and one-trial inhibitory avoidance was **enhanced** by either pre- or posttraining injections of **amphetamine** as well as 4-OH **amphetamine** and epinephrine. The finding that injections of **amphetamine** and epinephrine have comparable effects on **memory** is consistent with the view that **amphetamine** may modulate **memory** storage, at least in part, by inducing the release of epinephrine from the adrenal medulla. Metyrapone pretreatment blocked the **memory-enhancing** effects of **amphetamine**, 4-OH **amphetamine**, and epinephrine but did not affect retention performance of otherwise untreated animals. Posttraining injections of different doses of epinephrine (ranging from 0.0001 to 1.0 mg/kg) produced a dose-dependent **memory enhancement** for inhibitory avoidance training and metyrapone blocked the **memory-enhancing** effects of all these doses. These findings provide further evidence that the sympathoadrenal and adrenocortical systems are intimately coupled during processes of **memory** storage.
CT Check Tags: Animal; Comparative Study; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Adrenal Cortex: EN, enzymology
*Adrenal Cortex: SE, secretion
Adrenal Medulla: SE, secretion
***Amphetamine: PD, pharmacology**
Avoidance Learning: DE, drug effects
*Avoidance Learning: PH, physiology
*Corticosterone: PH, physiology
Depression, Chemical
***Epinephrine: PD, pharmacology**
Epinephrine: SE, secretion
*Metyrapone: PD, pharmacology
Rats
Rats, Sprague-Dawley
Retention (Psychology): DE, drug effects
Retention (Psychology): PH, physiology
*Steroid 11 beta-Monooxygenase: AI, antagonists & inhibitors
Stress, Psychological: PX, psychology
***p-Hydroxyamphetamine: PD, pharmacology**
RN **103-86-6 (p-Hydroxyamphetamine); 300-62-9 (Amphetamine)** ; 50-22-6 (Corticosterone); 51-43-4 (Epinephrine); 54-36-4 (Metyrapone)
CN EC 1.14.15.4 (Steroid 11 beta-Monooxygenase)

L111 ANSWER 4 OF 34 MEDLINE
AN **95388778** MEDLINE
DN **95388778** PubMed ID: **7659762**
TI Effect of **amphetamine** on long-term retention of verbal material.
AU Soetens E; Casaer S; D'Hooge R; Hueting J E
CS Laboratory of Experimental Psychology, University of Brussels, Belgium.
SO PSYCHOPHARMACOLOGY, **(1995 May)** 119 (2) 155-62.
Journal code: 7608025. ISSN: 0033-3158.
CY GERMANY: Germany, Federal Republic of
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Priority Journals
EM 199510
ED Entered STN: 19951013
Last Updated on STN: 19980206
Entered Medline: 19951003
AB A series of five experiments was conducted to investigate the temporal aspects of human **memory** consolidation of symbolic material through the administration of **amphetamine**. Subjects had to **recall** or recognise unrelated words from a previously presented list. The first experiments support the conjecture, based on animal studies, that **amphetamine enhances** long-term **memory** performance. Subsequently, **enhancement** is demonstrated with oral administration before learning, as well as with intramuscular injection after learning. It is shown that improved **recall** cannot be explained solely by general arousal or attentional processes, but must be due to consolidation. By introducing different test delays we show that consolidation of symbolic material can be modulated by **amphetamine** during the 1st hour after learning. In a final experiment we demonstrate that the **memory enhancement** applies to **recall** as well as to recognition. The implications of the present results are discussed in the context of recent research on LTP processes.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't
Administration, Oral
Adult
***Amphetamine: PD, pharmacology**
Double-Blind Method
Long-Term Potentiation: DE, drug effects

***Memory: DE, drug effects**
Mice
Recall: DE, drug effects
Retention (Psychology): DE, drug effects
RN **300-62-9 (Amphetamine)**

L111 ANSWER 5 OF 34 MEDLINE
AN **95346327** MEDLINE
DN **95346327** PubMed ID: **7620915**
TI **Amphetamine enhances memory** retention and facilitates norepinephrine release from the hippocampus in rats.
AU Lee E H; Ma Y L
CS Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan, The Republic of China.
SO BRAIN RESEARCH BULLETIN, **(1995)** 37 (4) 411-6.
Journal code: 7605818. ISSN: 0361-9230.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199508
ED Entered STN: 19950911
Last Updated on STN: 19950911
Entered Medline: 19950830
AB The present study investigated the effects of intrahippocampal **amphetamine** on **memory** retention and the role of hippocampal norepinephrine (NE) in **memory** consolidation in rats. One-way inhibitory avoidance learning paradigm was adopted. Animals were trained to avoid the foot shock. The latency to step into the shock compartment was recorded as the retention measure. The ceiling score (full retention) was 600 s. Results indicated that intra-hippocampal injections of **amphetamine** produced a dose-dependent **enhancement** of **memory** retention with doses at 0.6 micrograms and 1.6 micrograms reaching a significant effect. The beta-adrenergic blocker propranolol, at a dose which did not affect retention alone (80 ng), antagonized the **memory-enhancing** effect of **amphetamine**. Along with this **memory-enhancing** effect, **amphetamine** also elevated the level of NE release, and this effect was significant in animals not showing a full retention score (nonresponders) than in animals showing a full retention score (responders), as assayed by in vivo microdialysis. Within the control group, the responders also had a higher level of NE than the nonresponders. All these results are probably due to the fact that responders have a higher level of NE release than nonresponders. The effect of **amphetamine** on NE release is, therefore, not as obvious in responders. These results **together** support our hypothesis that NE plays a facilitatory role in the **memory** process and **amphetamine enhances** retention performance, at least in part, through facilitation of hippocampal NE release.
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
Amphetamine: AD, administration & dosage
***Amphetamine: PD, pharmacology**
Avoidance Learning: DE, drug effects
Dose-Response Relationship, Drug
Hippocampus: AH, anatomy & histology
Hippocampus: DE, drug effects
*Hippocampus: ME, metabolism
Injections
***Memory: DE, drug effects**
Microdialysis
Motor Activity: DE, drug effects
***Norepinephrine: ME, metabolism**

Rats
Rats, Sprague-Dawley
Receptors, Adrenergic: DE, drug effects
Stimulation, Chemical
RN **300-62-9 (Amphetamine)**; 51-41-2 (Norepinephrine)
CN 0 (Receptors, Adrenergic)

L111 ANSWER 6 OF 34 MEDLINE
AN **94077486** MEDLINE
DN **94077486** PubMed ID: **8255556**
TI **Amphetamine enhances** human-**memory** consolidation.
AU Soetens E; D'Hooge R; Hueting J E
CS Laboratory of Experimental Psychology, University of Brussels, Belgium.
SO NEUROSCIENCE LETTERS, **(1993 Oct 14)** 161 (1) 9-12.
Journal code: 7600130. ISSN: 0304-3940.
CY Ireland
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Priority Journals
EM 199401
ED Entered STN: 19940203
Last Updated on STN: 19940203
Entered Medline: 19940107
AB Although it is generally accepted that CNS stimulants have **enhancing** effects on long-term storage processes in laboratory animals, little is known about their influence on human learning. We report a series of experiments with free **recall** of lists of unrelated words, demonstrating a significant **enhancement** on long-term retention after **amphetamine** administration. A gradual increase of **recall** was observed up to 1 h after learning, remaining stable for at least 3 days, after oral administration before learning as well as intramuscular injection after learning. The results show that research on humans with drug-induced **memory-enhancement** techniques is necessary to supplement the animal studies for the understanding of the mechanisms involved in information consolidation.
CT Check Tags: Human; Male
***Amphetamine: PD, pharmacology**
Double-Blind Method
Learning: DE, drug effects
***Memory: DE, drug effects**
Placebos
RN **300-62-9 (Amphetamine)**
CN 0 (Placebos)

L111 ANSWER 7 OF 34 MEDLINE
AN **92279378** MEDLINE
DN **92279378** PubMed ID: **1594652**
TI Cocaine and **amphetamine** facilitate retention of jump-up responding in rats.
AU Janak P H; Martinez J L Jr
CS Department of Psychology, University of California, Berkeley 94720.
NC DA05375 (NIDA)
DA06192 (NIDA)
SO PHARMACOLOGY, BIOCHEMISTRY AND BEHAVIOR, **(1992 Apr)** 41 (4) 837-40.
Journal code: 0367050. ISSN: 0091-3057.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199206
ED Entered STN: 19920710
Last Updated on STN: 19920710
Entered Medline: 19920630
AB The effects of cocaine and **d-amphetamine** administration on the acquisition of an automated jump-up active avoidance task were examined in two separate experiments. On days 1 and 2, male Sprague-Dawley rats received one escape-only training trial, followed immediately by the intraperitoneal injection of cocaine, **amphetamine**, or saline. On day 3, subjects received eight escape/avoidance trials. The posttraining administration of cocaine (2.75 and 5.55 mg/kg) and **amphetamine** (0.3 and 1.0 mg/kg) on days 1 and 2 facilitated jump-up avoidance performance on day 3. Importantly, both cocaine and **amphetamine enhanced** learning and **memory** under experimental conditions that allowed for drug-free training and testing.
CT Check Tags: Animal; Male; Support, U.S. Gov't, P.H.S.
Amphetamine: AD, administration & dosage
***Amphetamine: PD, pharmacology**
*Avoidance Learning: DE, drug effects
Cocaine: AD, administration & dosage
***Cocaine: PD, pharmacology**
***Memory: DE, drug effects**
Rats
Rats, Inbred Strains
RN **300-62-9 (Amphetamine)**; 50-36-2 (Cocaine)

L111 ANSWER 8 OF 34 MEDLINE
AN **92239755** MEDLINE
DN **92239755** PubMed ID: **1810463**
TI Scopolamine **enhances** expression of an **amphetamine** -conditioned place preference.
AU Lynch M R
CS Research Serv-151, VA Medical Center, Syracuse, NY 13210.
SO NEUROREPORT, **(1991 Nov)** 2 (11) 715-8.
Journal code: 9100935. ISSN: 0959-4965.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199206
ED Entered STN: 19920619
Last Updated on STN: 19920619
Entered Medline: 19920603
AB Animals in the present investigation were trained for conditioned place preference by pairing the non-preferred compartment of a two chamber apparatus with either 1.5 mg kg-1 **D-amphetamine** or 0.05 mg kg-1 scopolamine. Some of the **amphetamine**-conditioned rats were injected with 0.05 mg kg-1 scopolamine as an acute treatment on the test day which followed conditioning. Although the scopolamine by itself did not induce either a preference or an aversion to the drug-paired side, it **enhanced** the expression of place preference in animals conditioned with **amphetamine. Potentiation** of this conditioned response (CR) was observed in the absence of changes in locomotor activation which would implicate general arousal as a potential mechanism. Hypotheses regarding anticholinergic mediation of CR expression via central reward mechanisms, **memory** retrieval, cue function and stimulus saliency are discussed, and possible neurosubstrates considered.
CT Check Tags: Animal; Male; Support, U.S. Gov't, Non-P.H.S.
Arousal: DE, drug effects
*Conditioning, Operant: DE, drug effects

***Dextroamphetamine: PD, pharmacology**
Dopamine: PH, physiology
Drug Synergism
Locomotion: DE, drug effects
Motivation
Rats
Rats, Inbred Strains
*Reward
***Scopolamine: PD, pharmacology**
*Spatial Behavior
Stimulation, Chemical
RN 51-34-3 (Scopolamine); 51-61-6 (Dopamine); **51-64-9 (Dextroamphetamine)**

L111 ANSWER 9 OF 34 MEDLINE
AN **91328728** MEDLINE
DN **91328728** PubMed ID: **1867627**
TI Time-dependent effects of post-trial **amphetamine** treatment in rats: evidence for **enhanced** storage of representational **memory.**
AU Strupp B J; Bunsey M; Levitsky D; Kesler M
CS Division of Nutritional Sciences, Cornell University, Ithaca, New York 14853.
NC NS20345 (NINDS)
SO BEHAVIORAL AND NEURAL BIOLOGY, **(1991 Jul)** 56 (1) 62-76.
Journal code: 7905471. ISSN: 0163-1047.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199109
ED Entered STN: 19910929
Last Updated on STN: 19910929
Entered Medline: 19910912
AB Two studies were conducted to test the ability of post-trial **amphetamine** treatment to improve later **recall** in a nonaversively motivated task. These studies utilized 8- and 12-arm radial mazes, respectively, with an 11-h retention interval imposed after the rat traversed half the arms of the maze (termed, the to-be-remembered-event, or TBRE). In Experiment 1, the rats were injected with **amphetamine** (0, .25, and .50 mg/kg) immediately after the TBRE. Because the drug treatment improved retention, a time dependency study was conducted in which the drug (0 and .33 mg/kg) was administered 0, 3, and 6.h after the TBRE. The finding that **amphetamine** injection at 0, but not 3, h post-trial improved later **recall** indicates that the benefit derived from the former treatment is not due to proactive influences at the time of the retention test. Drug treatment 6 h post-trial produced a borderline improvement of **recall**; possible mechanisms are discussed. Two conclusions can be drawn from these results: (1) **amphetamine** administration can improve **recall** under conditions in which this effect cannot be attributed to alterations in information processing during either the learning or the retention sessions, indicating that the drug modulates **memory** storage processes; and (2) **amphetamine** treatment can improve working **memory**, thus excluding an alternative interpretation for the previous reports of **impaired** short-term **memory** in animals, all of which entailed assessments of working **memory.** The possibility remains, however, that the **impairment** seen in these tasks reflects the requirement for erasure of information from previous trials within each daily session, rather than the duration of the retention interval.
CT Check Tags: Animal; Male; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

***Amphetamine: PD, pharmacology**
*Appetitive Behavior: DE, drug effects
*Discrimination Learning: DE, drug effects
Dose-Response Relationship, Drug
Injections, Subcutaneous
Motivation
*Orientation: DE, drug effects
Rats
***Recall: DE, drug effects**
***Retention (Psychology): DE, drug effects**
Time Factors
RN **300-62-9 (Amphetamine)**

L111 ANSWER 10 OF 34. MEDLINE
AN 91083132 MEDLINE
DN 91083132 PubMed ID: 1984711
TI Cognitive and behavioral effects of the **coadministration** of **dextroamphetamine** and haloperidol in schizophrenia.
AU Goldberg T E; Bigelow L B; Weinberger D R; Daniel D G; Kleinman J E
CS Clinical Brain Disorders Branch, NIMH Neurosciences Center at St. Elizabeths, Washington, DC 20032.
SO AMERICAN JOURNAL OF PSYCHIATRY, **(1991 Jan)** 148 (1) 78-84.
Journal code: 0370512. ISSN: 0002-953X.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199101
ED Entered STN: 19910322
Last Updated on STN: 19910322
Entered Medline: 19910128
AB OBJECTIVE: The authors sought to determine if an acute dose of **dextroamphetamine** might have positive effects on affect and cognition in schizophrenic patients maintained on a regimen of haloperidol and, if so, what variables might predict such improvements. METHOD: Twenty-one patients with chronic schizophrenia who were hospitalized on a research ward received a single oral dose of **dextroamphetamine** (0.25 mg/kg) in a double-blind, placebo-controlled, crossover study. All patients were receiving 0.4 mg/kg per day of haloperidol. Cognitive tests, motor tests, global ratings, mood ratings, and videotape ratings were used to determine the effect of the **coadministration** of these drugs. Ventricle-brain ratios derived from CT scans were used to predict response to the **coadministration** of these drugs. RESULTS: **Amphetamine** improved performance on a measure of concept formation on the Wisconsin Card Sorting Test but did not result in changes in performance on tests of **memory** or attention. As a group, the patients were more active and performed psychomotor tests more quickly while receiving **amphetamine**. Six patients were judged by clinical raters to have improved in terms of affect, cooperation, and engagement with the environment. Improvement was associated with enlarged cerebral ventricles and increases in blink rate from the placebo to the active drug condition. No patient unequivocally worsened. CONCLUSIONS: These results may be consistent with the theory that **coadministration** of **amphetamine** and haloperidol produces relatively selective **enhancement** of cortical dopaminergic activity. However, because of the acute nature of the trial and the specialized research environment in which it was conducted, the authors do not advocate **amphetamine** as a routine clinical treatment of schizophrenia.
CT Check Tags: Comparative Study; Female; Human; Male
Adult

Affect: DE, drug effects
Blinking: DE, drug effects
Cerebral Ventricles: AH, anatomy & histology
Chronic Disease
*Cognition: DE, drug effects
Concept Formation: DE, drug effects
Dextroamphetamine: AD, administration & dosage
Dextroamphetamine: PD, pharmacology
***Dextroamphetamine: TU, therapeutic use**
Double-Blind Method
Drug Therapy, Combination
Haloperidol: AD, administration & dosage
Haloperidol: PD, pharmacology
***Haloperidol: TU, therapeutic use**
Hospitalization
Middle Age
Psychological Tests
Schizophrenia: DI, diagnosis
*Schizophrenia: DT, drug therapy
Schizophrenia: RA, radiography
*Schizophrenic Psychology
RN **51-64-9 (Dextroamphetamine)**; 52-86-8 (Haloperidol)

L111 ANSWER 11 OF 34 MEDLINE
AN **89193569** MEDLINE
DN **89193569** PubMed ID: **3240294**
TI Alterations in calmodulin content of rat brain areas after chronic application of haloperidol and **amphetamine**.
AU Popov N; Schulzeck S; Nuss D; Vopel A U; Jendrny C; Struy H; Matthies H
CS Institute of Pharmacology and Toxicology, Medical Academy, Magdeburg, GDR.
SO BIOMEDICA BIOCHIMICA ACTA, **(1988)** 47 (4-5) 435-41.
Journal code: 8304435. ISSN: 0232-766X.
CY GERMANY, EAST: German Democratic Republic
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198904
ED Entered STN: 19900306
Last Updated on STN: 19900306
Entered Medline: 19890425
AB The water-soluble (cytosolic) and Lubrol-soluble (membrane-bound) calmodulin contents were determined radioimmunologically in fractions of striatum, hippocampus and cerebellum of dopamine supersensitive rats. Development of supersensitivity was the sequel of 3-weeks treatment of the animals with 1 mg/kg haloperidol or 5 mg/kg **amphetamine** i.p. daily. In the dopamine-rich striatum, the membrane-bound calmodulin content was increased by both modes of treatment, consistent with data from the literature. The patterns suggest that additional calmodulin was synthesized under the conditions studied. The hippocampus, the region poor in dopamine while playing an essential role in learning and **memory** formation processes, revealed similar patterns after both modes of treatment. However, in this region a pronounced translocation was seen, i.e. a redistribution from the cytosolic into the membrane compartment, without signs evidencing **enhanced** synthesis. The third region under investigation, the cerebellum, did not show any alterations in calmodulin content. Differentiation between pre- and postsynaptic changes was not possible. The results are discussed in the light of the present knowledge about participation of dopaminergic systems in processes of neuronal plasticity.
CT Check Tags: Animal; Male
***Amphetamine: PD, pharmacology**
Brain: DE, drug effects
*Brain: ME, metabolism

*Calmodulin: PD, pharmacology
Cytosol: ME, metabolism
***Haloperidol: PD, pharmacology**
Membranes: ME, metabolism
Organ Specificity
Rats
Rats, Inbred Strains
Reference Values
RN **300-62-9 (Amphetamine)**; 52-86-8 (Haloperidol)
CN 0 (Calmodulin)

L111 ANSWER 12 OF 34 MEDLINE
AN **89099378** MEDLINE
DN **89099378** PubMed ID: **3212062**
TI **Amphetamine enhances** retrieval following diverse sources of forgetting.
AU Quartermain D; Judge M E; Jung H
CS Department of Neurology, New York University School of Medicine.
NC MH 37326 (NIMH)
SO PHYSIOLOGY AND BEHAVIOR, **(1988)** 43 (2) 239-41.
Journal code: 0151504. ISSN: 0031-9384.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198902
ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19890221
AB The generality of **amphetamine**-induced retrieval **enhancement** was investigated by determining whether pretest administration could alleviate different types of forgetting. Thirsty mice were punished for licking a water tube following a period of free drinking. Forgetting of the conditioned drink suppression was induced in different groups of animals by; protein synthesis inhibition, cholinergic receptor blockade, inhibition of norepinephrine synthesis, stimulation of serotonin receptors, electroconvulsive shock, a 2.5 month training to test interval and the use of senescent animals with an endogenous **memory** defect. Thirty min prior to testing mice were injected with either saline or with 2 mg/kg **d-amphetamine** sulphate. Results showed that **amphetamine** produced a highly significant improvement in remembering in all of the forgetting treatment groups. It is concluded that **amphetamine** can alleviate forgetting caused by widely diverse etiologies probably by activating a nonspecific general retrieval system.
CT Check Tags: Animal; Male; Support, U.S. Gov't, P.H.S.
Amnesia
*Avoidance Learning
***Dextroamphetamine: PD, pharmacology**
Electroshock
***Memory: DE, drug effects**
Mice
Reference Values
RN **51-64-9 (Dextroamphetamine)**

L111 ANSWER 13 OF 34 MEDLINE
AN **88320725** MEDLINE
DN **88320725** PubMed ID: **3413232**
TI **d-Amphetamine enhances memory** performance in rats with damage to the fimbria.
AU M'Harzi M; Willig F; Costa J C; Delacour J
CS Laboratoire de Psychophysiologie, Universite Paris VII, France.
SO PHYSIOLOGY AND BEHAVIOR, **(1988)** 42 (6) 575-9.

Journal code: 0151504. ISSN: 0031-9384.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198810
ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19881012
AB Rats were preoperatively trained on a 5-unit linear maze and were then subjected to fimbria lesions. The animals were then retested on the same task with one group of rats with fimbria lesions and a control group being injected daily with 0.5 mg/kg **d-amphetamine** sulfate prior to testing. Lesions significantly **impaired** postoperative performance of the task, while **amphetamine** facilitated performance in fimbria lesioned rats. Due to an optimal learning of the task, performance of control animals was not significantly facilitated. These results raise several important issues including the mechanisms of functional recovery after brain lesions and the role of the hippocampal formation in learning and **memory**.
CT Check Tags: Animal; Male
***Dextroamphetamine: PD, pharmacology**
Hippocampus: IN, injuries
*Hippocampus: PH, physiology
Learning
***Memory: DE, drug effects**
Rats
Rats, Inbred Strains
RN **51-64-9 (Dextroamphetamine)**

L111 ANSWER 14 OF 34 MEDLINE
AN 88268672 MEDLINE
DN 88268672 PubMed ID: 3390096
TI Effects of scopolamine and **dextroamphetamine** on human performance.
AU Schmedtje J F Jr; Oman C M; Letz R; Baker E L
CS Man-Vehicle Laboratory, Massachusetts Institute of Technology, Cambridge.
SO AVIATION SPACE AND ENVIRONMENTAL MEDICINE, **(1988 May)** 59 (5) 407-10.
Journal code: 7501714. ISSN: 0095-6562.
Report No.: NASA-88268672.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 198807
ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880729
AB The effects of two drugs used to prevent symptoms of motion sickness in the operational environment were examined in this study of human performance as measured by computer-based tests of cognitive and psychomotor skills. Each subject was exposed repetitively to five tests: Symbol-Digit Substitution, Simple Reaction Time, Pattern Recognition, Digit Span **Memory**, and Pattern **Memory**. Although there have been previous reports of decreases in human performance in similar testing with higher dosages of scopolamine or **dextroamphetamine**, no significant decrements were observed with the operational-level **combined** dose used in this study (0.4 mg oral scopolamine and 5.0 mg oral **dextroamphetamine**.) The controversy over the use of **combination** drug therapy in this environment is discussed along with the indications for further research based on the findings.
CT Check Tags: Human; Support, U.S. Gov't, Non-P.H.S.

Attention
*Cognition: DE, drug effects
***Dextroamphetamine: AE, adverse effects**
Dextroamphetamine: TU, therapeutic use
Drug Therapy, Combination
Memory
Motion Sickness: DT, drug therapy
Pattern Recognition
*Psychomotor Performance: DE, drug effects
***Scopolamine: AE, adverse effects**
Scopolamine: TU, therapeutic use
Wechsler Scales
RN 51-34-3 (Scopolamine); **51-64-9 (Dextroamphetamine)**

L111 ANSWER 15 OF 34 MEDLINE
AN 86068658 MEDLINE
DN 86068658 PubMed ID: 4157252
TI [Treatment of psychopathologic sequelae of early childhood brain damage]. Behandlung der psychopathologischen Folgen fruhkindlicher Hirnschadigung.
AU Sulestrowska H
SO PSYCHIATRIE, NEUROLOGIE UND MEDIZINISCHE PSYCHOLOGIE. BEIHEFTE, **(1968)** 8-9 143-8.
Journal code: 0125315. ISSN: 0555-5469.
CY GERMANY, EAST: German Democratic Republic
DT Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 198601
ED Entered STN: 19900321
Last Updated on STN: 19950206
Entered Medline: 19860122
AB The pharmacotherapy of the psychopathological consequences of damages to the brain suffered in early childhood (erethistic or torpid oligophrenia, characteropathy, episodic psychic disorders in epilepsy, tics, and schizophrenic syndromes in encephalopathy) is discussed.
CT Check Tags: Human
Amphetamine: TU, therapeutic use
Antipsychotic Agents: TU, therapeutic use
*Brain Damage, Chronic: DT, drug therapy
Child
***Delirium, Dementia, Amnestic, Cognitive Disorders: DT, drug therapy**
Drug Therapy, Combination
English Abstract
Glutamates: TU, therapeutic use
Long-Term Care
Mental Retardation: DT, drug therapy
RN **300-62-9 (Amphetamine)**
CN 0 (Antipsychotic Agents); 0 (Glutamates)

L111 ANSWER 16 OF 34 MEDLINE
AN **84258537** MEDLINE
DN **84258537** PubMed ID: **6744050**
TI Modulation of long-term **potentiation** by peripherally administered **amphetamine** and epinephrine.
AU Gold P E; Delanoy R L; Merrin J
NC AG 01643 (NIA)
MH 31141 (NIMH)
SO BRAIN RESEARCH, **(1984 Jul 2)** 305 (1) 103-7.
Journal code: 0045503. ISSN: 0006-8993.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 198409
ED Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19840914
AB Long-term **potentiation** (LTP) has received considerable attention as a neurophysiological model for studying the biology of **memory**. The present experiments examined the susceptibility of LTP in the dentate gyrus to modification by peripheral injections of **amphetamine** and epinephrine. Both drugs **enhanced** the development of LTP in a dose-related manner comparable to that seen previously in behavioral studies. Such results suggest that the development of this long-lasting electrophysiological change can be regulated by peripheral catecholamine levels in a manner analogous to that seen in behavioral studies of **memory**.
CT Check Tags: Animal; Male; Support, U.S. Gov't, P.H.S.
***Amphetamine: PD, pharmacology**
Dose-Response Relationship, Drug
***Epinephrine: PD, pharmacology**
*Evoked Potentials: DE, drug effects
*Hippocampus: DE, drug effects
Memory: PH, physiology
Rats
Rats, Inbred Strains
Stimulation, Chemical
Sympathetic Nervous System: PH, physiology
Time Factors
RN **300-62-9 (Amphetamine)**; 51-43-4 (Epinephrine)

L111 ANSWER 17 OF 34 MEDLINE
AN **83230592** MEDLINE
DN **83230592** PubMed ID: **7183311**
TI **Memory** retrieval **enhanced** by **amphetamine** after a long retention interval.
AU Sara S J; Deweer B
SO BEHAVIORAL AND NEURAL BIOLOGY, **(1982 Oct)** 36 (2) 146-60. Journal code: 7905471. ISSN: 0163-1047.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198307
ED Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19830708
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
Appetitive Behavior: DE, drug effects
Conditioning, Operant: DE, drug effects
***Dextroamphetamine: PD, pharmacology**
*Discrimination Learning: DE, drug effects
Dose-Response Relationship, Drug
***Memory: DE, drug effects**
Motor Activity: DE, drug effects
Rats
Rats, Inbred Strains
***Recall: DE, drug effects**
***Retention (Psychology): DE, drug effects**
RN **51-64-9 (Dextroamphetamine)**

L111 ANSWER 18 OF 34 MEDLINE
AN **83170455** MEDLINE
DN **83170455** PubMed ID: **6403964**
TI Effect of naloxone and **amphetamine** on acquisition and

memory consolidation of active avoidance responses in rats.
AU Fulginiti S; Cancela L M
SO PSYCHOPHARMACOLOGY, **(1983)** 79 (1) 45-8.
Journal code: 7608025. ISSN: 0033-3158.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198305
ED Entered STN: 19900318
Last Updated on STN: 19900318
Entered Medline: 19830505
AB Pretraining IP injection of naloxone (0.3 mg/kg) or **amphetamine** (2 mg/kg) **enhanced** performance during acquisition, but did not improve retention of active avoidance responses in rats. Naloxone (0.1 or 3 mg/kg) had no effect on acquisition or on retention. The **combination** of naloxone (0.3 mg/kg) plus **amphetamine** (2 mg/kg) did not produce the facilitation observed when each of the two drugs was administered alone. Pretreatment with the higher dose of naloxone (3 mg/kg) blocked the facilitative effect of **amphetamine** on acquisition. Post-training administration of naloxone (0.3 mg/kg) or **amphetamine** (2 mg/kg) improved retention. Naloxone (0.1 or 3 mg/kg) had no effect. When naloxone and **amphetamine** were **combined**, at respective doses of 0.3 mg/kg and 2 mg/kg, the improvement did not occur, i.e., the higher dose of naloxone prevented the facilitative effect of **amphetamine**. In addition, an ineffective dose of **amphetamine** (0.5 mg/kg), given either pre- or post-training **together** with the lower dose of naloxone (0.1 mg/kg), produced a significant **enhancement** of acquisition or consolidation, respectively. The results are consistent with the possibility that naloxone might exert its facilitative action on acquisition and **memory** consolidation through the release of catecholaminergic systems from inhibitory influences of opioids.
CT Check Tags: Animal; Female; Support, Non-U.S. Gov't
***Amphetamine: PD, pharmacology**
*Avoidance Learning: DE, drug effects
Catecholamines: PH, physiology
***Memory: DE, drug effects**
*Naloxone: PD, pharmacology
Rats
Rats, Inbred Strains
RN **300-62-9 (Amphetamine)**; 465-65-6 (Naloxone)
CN 0 (Catecholamines)

L111 ANSWER 19 OF 34 MEDLINE
AN **83144600** MEDLINE
DN **83144600** PubMed ID: **6828532**
TI **Amphetamine** effects on long term **potentiation** in dentate granule cells.
AU Delanoy R L; Tucci D L; Gold P E
SO PHARMACOLOGY, BIOCHEMISTRY AND BEHAVIOR, **(1983 Jan)** 18 (1) 137-9.
Journal code: 0367050. ISSN: 0091-3057.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198304
ED Entered STN: 19900318
Last Updated on STN: 19900318
Entered Medline: 19830421
AB Long term **potentiation** (LTP) has received considerable attention as a neurophysiological analog of **memory**. **Amphetamine**,

as well as several other catecholamine agonists, can **enhance** behaviorally-assessed **memory** storage in a variety of training situations. The present experiments tested the effects of **amphetamine** on LTP produced by high frequency stimulation of the perforant path in rats. The results indicate that **amphetamine** can **enhance** the development of LTP under some but not all testing procedures. Studies of the neurobiological bases by which central and peripheral catecholamines modulate **memory** storage may be augmented by examinations of catecholamine effects on a specific form of long-lasting change in brain function. Similarly, the ability to manipulate LTP may prove to be an important aid in examinations of neurobiological correlates of this phenomenon.

CT Check Tags: Animal; Male
***Amphetamine: PD, pharmacology**
Electric Stimulation
Evoked Potentials: DE, drug effects
Hippocampus: DE, drug effects
*Hippocampus: PH, physiology
***Memory: DE, drug effects**
Rats
Rats, Inbred Strains

RN **300-62-9 (Amphetamine)**

L111 ANSWER 20 OF 34 MEDLINE
AN **82127800** MEDLINE
DN **82127800** PubMed ID: **6949168**
TI Acquisition and retrieval of information in **amphetamine**-treated hyperactive children.
AU Weingartner H; Langer D; Grice J; Rapoport J L
SO PSYCHIATRY RESEARCH, **(1982 Feb)** 6 (1) 21-9.
Journal code: 7911385. ISSN: 0165-1781.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198204
ED Entered STN: 19900317
Last Updated on STN: 19970203
Entered Medline: 19820422

AB State-dependent learning and **memory** (retrieval) processes were examined in 15 **amphetamine**-treated hyperactive boys. While stimulant treatment **enhanced** the acquisition of information and its retrieval 24 hours later, there was no evidence of poorer retrieval of information learned in a state different from the retrieval state. **Amphetamine** appeared particularly to facilitate effortful cognitive processes. Subgroups of hyperactive children respond to **amphetamine** treatment in different ways, some showing changes in motor restlessness and others changes in cognition. The lack of dissociative effects when information is learned and **recalled** under different drug conditions suggests that what the stimulant-treated child learns can be effectively recovered after completion of treatment.

CT Check Tags: Human; Male
Attention: DE, drug effects
*Attention Deficit Disorder with Hyperactivity: DT, drug therapy
Attention Deficit Disorder with Hyperactivity: PX, psychology
Child
*Concept Formation: DE, drug effects
***Dextroamphetamine: TU, therapeutic use**
*Learning Disorders: DT, drug therapy
Learning Disorders: PX, psychology
***Memory: DE, drug effects**
***Recall: DE, drug effects**
Serial Learning: DE, drug effects

Verbal Learning: DE, drug effects
RN **51-64-9 (Dextroamphetamine)**

L111 ANSWER 21 OF 34 MEDLINE
AN **82082808** MEDLINE
DN **82082808** PubMed ID: **7312905**
TI Short-term **memory**: the role of **d-amphetamine**
.
AU Kesner R P; Bierley R A; Pebbles P
NC RR07092-12 (NCRR)
SO PHARMACOLOGY, BIOCHEMISTRY AND BEHAVIOR, **(1981 Nov)** 15 (5) 673-6.
Journal code: 0367050. ISSN: 0091-3057.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198202
ED Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19820212
AB **d-Amphetamine** injections produce a dose-dependent disruption of performance within a discrete delayed alternation and a spatial delayed matching-to-sample task. Since **d-amphetamine** in the doses used had no deleterious effects on discrimination performance (no delay condition), it is suggested that **d-amphetamine** disrupts neuronal activity representing short-term **memory**. The data provide support for an independence model of short- and long-term **memory**.
CT Check Tags: Animal; Male; Support, U.S. Gov't, P.H.S.
Conditioning, Operant: DE, drug effects
***Dextroamphetamine: PD, pharmacology**
***Memory, Short-Term: DE, drug effects**
Motor Activity: DE, drug effects
Rats
RN **51-64-9 (Dextroamphetamine)**

L111 ANSWER 22 OF 34 MEDLINE
AN **80240667** MEDLINE
DN **80240667** PubMed ID: **6994586**
TI **Memory enhancement** in **Korsakoff**'s psychosis by clonidine: further evidence for a noradrenergic deficit.
AU McEntee W J; Mair R G
SO ANNALS OF NEUROLOGY, **(1980 May)** 7 (5) 466-70.
Journal code: 7707449. ISSN: 0364-5134.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198009
ED Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800928
AB Three drugs, **d-amphetamine**, clonidine, and methysertide, which presumably **enhance** central noradrenergic activity by different pharmacological mechanisms, were administered to eight patients with the **Korsakoff** syndrome in a two-week subacute, double-blind, counterbalanced experiment to study the effects of these agents on **memory** function as measured by a neuropsychological test battery. Of the drugs tested, only clonidine, a putative alpha-noradrenergic agonist, was associated with significant improvement in **memory**. The data are consistent with the

hypothesis that damage to ascending norepinephrine-containing neurons in the brainstem and diencephalon may be the basis for **amnesia** in **Korsakoff**'s psychosis.
CT Check Tags: Human; Support, U.S. Gov't, Non-P.H.S.
Adult
***Alcohol Amnestic Disorder: DT, drug therapy**
Alcohol Amnestic Disorder: PP, physiopathology
Clinical Trials
***Clonidine: TU, therapeutic use**
***Dextroamphetamine: TU, therapeutic use**
Double-Blind Method
Memory: PH, physiology
*Methysergide: TU, therapeutic use
Middle Age
Neural Pathways: PP, physiopathology
Norepinephrine: PH, physiology
RN 361-37-5 (Methysergide); 4205-90-7 (Clonidine); 51-41-2 (Norepinephrine); **51-64-9 (Dextroamphetamine)**

L111 ANSWER 23 OF 34 MEDLINE
AN **80089423** MEDLINE
DN **80089423** PubMed ID: **7350983**
TI Central and peripheral actions of **amphetamine** on **memory** storage.
AU Martinez J L Jr; Jensen R A; Messing R B; Vasquez B J; Soumireu-Mourat B; Geddes D; Liang K C; McGaugh J L
SO BRAIN RESEARCH, **(1980 Jan 20)** 182 (1) 157-66.
Journal code: 0045503. ISSN: 0006-8993.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198003
ED Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800317
AB These experiments investigated the effects of central (intracerebroventricular) and peripheral (i.p.) posttraining administration of **D-amphetamine** on rat's retention of a one-trial inhibitory avoidance response. While retention was **enhanced** by i.p. administration (1.0 mg/kg) the central administration (dose range 50-500 microgram) did not affect retention. In rats given peripheral 6-OHDA 24 h prior to training a lower dose (i.p.) of **amphetamine** (0.25 mg/kg) was most effective in **enhancing** retention. These findings suggest that the mrmory **enhancing** effects of **D-amphetamine** are mediated at least in part through peripheral systems.
CT Check Tags: Animal; Male; Support, U.S. Gov't, P.H.S.
Avoidance Learning: DE, drug effects
***Dextroamphetamine: PD, pharmacology**
Dose-Response Relationship, Drug
Hydroxydopamines: PD, pharmacology
Injections, Intraventricular
***Memory: DE, drug effects**
Motor Activity: DE, drug effects
Myocardium: ME, metabolism
Norepinephrine: ME, metabolism
Rats
***Retention (Psychology): DE, drug effects**
Sympathetic Nervous System: DE, drug effects
RN 51-41-2 (Norepinephrine); **51-64-9 (Dextroamphetamine)**
CN 0 (Hydroxydopamines)

L111 ANSWER 24 OF 34 MEDLINE
AN **78248979** MEDLINE
DN **78248979** PubMed ID: **684096**
TI A possible physiological mechanism for short-term **memory**.
AU Gibbs M E; Gibbs C L; Ng K T
SO PHYSIOLOGY AND BEHAVIOR, **(1978 May)** 20 (5) 619-27.
Journal code: 0151504. ISSN: 0031-9384.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197810
ED Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19781027
CT Check Tags: Animal; Male
*Animals, Newborn: PH, physiology
*Avoidance Learning: PH, physiology
Brain
Chickens
Dextroamphetamine: PD, pharmacology
Dose-Response Relationship, Drug
Extracellular Space: PH, physiology
Injections
***Memory, Short-Term: PH, physiology**
Phenytoin: PD, pharmacology
*Potassium: PH, physiology
Potassium Chloride: AD, administration & dosage
*Sodium: PH, physiology
Sodium Chloride: AD, administration & dosage
RN **51-64-9 (Dextroamphetamine)**; 57-41-0 (Phenytoin); 7440-09-7 (Potassium); 7440-23-5 (Sodium); 7447-40-7 (Potassium Chloride); 7647-14-5 (Sodium Chloride)

L111 ANSWER 25 OF 34 MEDLINE
AN 76170978 MEDLINE
DN 76170978 PubMed ID: 1262859
TI Treatment of chronic post-traumatic organic brain syndrome with **dextroamphetamine**: first reported case.
AU Lipper S; Tuchman M M
SO JOURNAL OF NERVOUS AND MENTAL DISEASE, **(1976 May)** 162 (5) 366-71.
Journal code: 0375402. ISSN: 0022-3018.
CY United States
DT (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197607
ED Entered STN: 19900313
Last Updated on STN: 19980206
Entered Medline: 19760706
AB In view of its therapeutic efficacy in the treatment of children with minimal brain dysfunction syndrome, **dextroamphetamine** was administered to a young adult with a chronic organic brain syndrome secondary to cerebral trauma. That **D-amphetamine** was critical to the resulting marked diminution in confusion, paranoia, and deficit in short term **memory** was confirmed by the occurrence of a relapse coincident with placebo administration as part of a double blind evaluation. Amitriptylline appeared to **potentiate** the therapeutic effects of **D-amphetamine**. The results achieved, although observational and subjective in nature, warrant

replication in controlled, quantitative clinical studies.
CT Check Tags: Case Report; Human; Male
Accidents, Traffic
Adult
Amitriptyline: AD, administration & dosage
Amitriptyline: TU, therapeutic use
*Brain Injuries: CO, complications
Chlorpromazine: AD, administration & dosage
Chlorpromazine: TU, therapeutic use
***Delirium, Dementia, Amnestic, Cognitive Disorders: DT, drug therapy**
Delirium, Dementia, Amnestic, Cognitive Disorders: ET, etiology
Dextroamphetamine: AD, administration & dosage
***Dextroamphetamine: TU, therapeutic use**
Drug Therapy, Combination
RN 50-48-6 (Amitriptyline); 50-53-3 (Chlorpromazine); **51-64-9 (Dextroamphetamine)**

L111 ANSWER 26 OF 34 MEDLINE
AN **75031182** MEDLINE
DN **75031182** PubMed ID: **4423372**
TI **d-Amphetamine** effects on attention and **memory** in the albino and hooded rat.
AU Beckwith B E; Sandman C A; Alexander W D; Gerald M C; Goldman H
SO PHARMACOLOGY, BIOCHEMISTRY AND BEHAVIOR, **(1974 Jul-Aug)** 2 (4) 557-61.
Journal code: 0367050. ISSN: 0091-3057.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197501
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750110
CT Check Tags: Animal; Male
Analysis of Variance
*Attention: DE, drug effects
Dextroamphetamine: AD, administration & dosage
***Dextroamphetamine: PD, pharmacology**
Discrimination Learning: DE, drug effects
***Memory: DE, drug effects**
Rats
Reversal Learning: DE, drug effects
Species Specificity
RN **51-64-9 (Dextroamphetamine)**

L111 ANSWER 27 OF 34 MEDLINE
AN **73259537** MEDLINE
DN **73259537** PubMed ID: **4581912**
TI Drug facilitation of learning and **memory**.
AU McGaugh J L
SO ANNUAL REVIEW OF PHARMACOLOGY, **(1973)** 13 229-41. Ref: 98
Journal code: 7607089. ISSN: 0066-4251.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 197311
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19731116

CT Check Tags: Animal
Amphetamine: PD, pharmacology
Bemegride: PD, pharmacology
Discrimination Learning: DE, drug effects
Guinea Pigs
*Learning: DE, drug effects
***Memory: DE, drug effects**
Nicotine: PD, pharmacology
Parasympathomimetics: PD, pharmacology
Pemoline
Pentylenetetrazole: PD, pharmacology
Picrotoxin: PD, pharmacology
RNA: PD, pharmacology
Rats
Strychnine: PD, pharmacology
Time Factors
RN 124-87-8 (Picrotoxin); 2152-34-3 (Pemoline); **300-62-9 (Amphetamine)**; 54-11-5 (Nicotine); 54-95-5 (Pentylenetetrazole); 57-24-9 (Strychnine); 63231-63-0 (RNA); 64-65-3 (Bemegride)
CN 0 (Parasympathomimetics)

L111 ANSWER 28 OF 34 MEDLINE
AN **73015532** MEDLINE
DN **73015532** PubMed ID: **4403945**
TI Drugs and **memory** disorders in human aging.
AU Jarvik M E; Gritz E R; Schneider N G
SO BEHAVIORAL BIOLOGY, **(1972 Oct)** 7 (5) 643-68. Ref: 78
Journal code: 0326100. ISSN: 0091-6773.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 197212
ED Entered STN: 19900310
Last Updated on STN: 19950206
Entered Medline: 19721204
CT Check Tags: Human
Adolescence
Adult
Aged
*Aging
Amphetamine: TU, therapeutic use
Anticonvulsants: TU, therapeutic use
Antidepressive Agents: TU, therapeutic use
Arousal: DE, drug effects
Caffeine: TU, therapeutic use
Central Nervous System Stimulants: PD, pharmacology
Central Nervous System Stimulants: TU, therapeutic use
Cerebrovascular Circulation
Child
Hallucinogens: TU, therapeutic use
Hyperbaric Oxygenation
Hypnotics and Sedatives: TU, therapeutic use
Learning: DE, drug effects
***Memory Disorders: DT, drug therapy**
Memory Disorders: TH, therapy
Middle Age
Nicotine: PD, pharmacology
Nutrition
Parasympathomimetics: PD, pharmacology
Procaine: TU, therapeutic use
Sympathomimetics: TU, therapeutic use

RN **300-62-9 (Amphetamine)**; 54-11-5 (Nicotine); 58-08-2 (Caffeine); 59-46-1 (Procaine)
CN 0 (Anticonvulsants); 0 (Antidepressive Agents); 0 (Central Nervous System Stimulants); 0 (Hallucinogens); 0 (Hypnotics and Sedatives); 0 (Parasympathomimetics); 0 (Sympathomimetics)

L111 ANSWER 29 OF 34 MEDLINE
AN **72257621** MEDLINE
DN **72257621** PubMed ID: **4949130**
TI Drug effects and learning and **memory** processes.
AU Essman W B
SO ADVANCES IN PHARMACOLOGY AND CHEMOTHERAPY, **(1971)** 9 241-330.
Ref: 248
Journal code: 0237113. ISSN: 0065-3144.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 197210
ED Entered STN: 19900310
Last Updated on STN: 19970203
Entered Medline: 19721005
CT Check Tags: Animal
Amines: PD, pharmacology
Amphetamine: PD, pharmacology
Caffeine: PD, pharmacology
Catecholamines: PD, pharmacology
Hypnotics and Sedatives: PD, pharmacology
Indoles: PD, pharmacology
*Learning: DE, drug effects
Macromolecular Systems
Magnesium
Malonates: PD, pharmacology
***Memory: DE, drug effects**
Nicotine: PD, pharmacology
Nitriles: PD, pharmacology
Parasympathetic Nervous System: DE, drug effects
Pemoline: PD, pharmacology
Pentylenetetrazole: PD, pharmacology
Picrotoxin: PD, pharmacology
RNA: PD, pharmacology
Strychnine: PD, pharmacology
Tranquilizing Agents: PD, pharmacology
Uric Acid: PD, pharmacology
RN 124-87-8 (Picrotoxin); 2152-34-3 (Pemoline); **300-62-9 (Amphetamine)**; 54-11-5 (Nicotine); 54-95-5 (Pentylenetetrazole); 57-24-9 (Strychnine); 58-08-2 (Caffeine); 63231-63-0 (RNA); 69-93-2 (Uric Acid); 7439-95-4 (Magnesium)
CN 0 (Amines); 0 (Catecholamines); 0 (Hypnotics and Sedatives); 0 (Indoles); 0 (Macromolecular Systems); 0 (Malonates); 0 (Nitriles); 0 (Tranquilizing Agents)

L111 ANSWER 30 OF 34 MEDLINE
AN **72161251** MEDLINE
DN **72161251** PubMed ID: **4259732**
TI Involvement of biogenic amines in **memory** formation.
AU Dismukes R K; Rake A V
SO PSYCHOPHARMACOLOGIA, **(1972)** 23 (1) 17-25.
Journal code: 7609417. ISSN: 0033-3158.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 197206
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19720622
CT Check Tags: Animal; Female; Male
5-Hydroxytryptophan: PD, pharmacology
Amphetamine: PD, pharmacology
*Avoidance Learning: DE, drug effects
Biogenic Amines: ME, metabolism
Brain: ME, metabolism
Brain Chemistry: DE, drug effects
*Catecholamines: ME, metabolism
Dihydroxyphenylalanine: PD, pharmacology
Dopamine: ME, metabolism
Epinephrine: ME, metabolism
Fenclonine: PD, pharmacology
***Memory: DE, drug effects**
Mice
Norepinephrine: ME, metabolism
***Reserpine: PD, pharmacology**
*Serotonin: ME, metabolism
Thiocarbamates: PD, pharmacology
RN **300-62-9 (Amphetamine)**; 50-55-5 (Reserpine); 50-67-9 (Serotonin); 51-41-2 (Norepinephrine); 51-43-4 (Epinephrine); 51-61-6 (Dopamine); 56-69-9 (5-Hydroxytryptophan); 63-84-3 (Dihydroxyphenylalanine); 7424-00-2 (Fenclonine)
CN 0 (Biogenic Amines); 0 (Catecholamines); 0 (Thiocarbamates)

L111 ANSWER 31 OF 34 MEDLINE
AN 72157310 MEDLINE
DN 72157310 PubMed ID: 5145597
TI **Amphetamine**-barbiturate **mixtures**: learning and retention in rats.
AU Porsolt R D; Joyce D; Summerfield A
SO ACTIVITAS NERVOSA SUPERIOR, **(1971)** 13 (2) 75-7.
Journal code: 0400662. ISSN: 0001-7604.
CY Czechoslovakia
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197206
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19720619
CT Check Tags: Animal; Comparative Study
***Amphetamine: PD, pharmacology**
*Barbiturates: PD, pharmacology
Drug Synergism
*Learning: DE, drug effects
***Memory: DE, drug effects**
Rats
Reinforcement (Psychology)
Reversal Learning: DE, drug effects
RN **300-62-9 (Amphetamine)**
CN 0 (Barbiturates)

L111 ANSWER 32 OF 34 MEDLINE
AN 72083082 MEDLINE
DN 72083082 PubMed ID: 5134295
TI Apparent delayed **enhancement** of **memory** following post-trial **methylamphetamine** hydrochloride.
AU Johnson F N; Waite K

SO EXPERIENTIA, **(1971)** 27 (11) 1316-7.
Journal code: 0376547. ISSN: 0014-4754.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197203
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19720320
CT Check Tags: Animal; Male
***Amphetamine: PD, pharmacology**
Avoidance Learning
Electroshock
Extinction (Psychology): DE, drug effects
***Memory: DE, drug effects**
Rats
Time Factors
RN **300-62-9 (Amphetamine)**

L111 ANSWER 33 OF 34 MEDLINE
AN **69028191** MEDLINE
DN **69028191** PubMed ID: **5246555**
TI Arousal and the conversion of "short-term" to "long-term" **memory**
.
AU Barondes S H; Cohen H D
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, **(1968 Nov)** 61 (3) 923-9.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196812
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19681220
CT Check Tags: Animal; Male
Amphetamine: PD, pharmacology
*Arousal
Brain Chemistry
Cycloheximide: PD, pharmacology
Drug Antagonism
Injections, Subcutaneous
***Memory: DE, drug effects**
Mice
Proteins: BI, biosynthesis
Time Factors
RN **300-62-9 (Amphetamine)**; 66-81-9 (Cycloheximide)
CN 0 (Proteins)

L111 ANSWER 34 OF 34 MEDLINE
AN **66005400** MEDLINE
DN **66005400** PubMed ID: **5318331**
TI Some effects of morphine and **amphetamine** on intellectual functions and mood.
AU Evans W O; Smith R P
SO PSYCHOPHARMACOLOGIA, **(1964 Jul 6)** 6 (1) 49-56.
Journal code: 7609417. ISSN: 0033-3158.
CY GERMANY, WEST: Germany, Federal Republic of
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 196511
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19651120
CT Check Tags: Comparative Study; Human
***Amphetamine: PD, pharmacology**
Clinical Trials
*Cognition
***Dextroamphetamine: PD, pharmacology**
***Memory**
*Mental Processes
*Morphine: PD, pharmacology
*Psychological Tests
*Thinking
RN **300-62-9 (Amphetamine); 51-64-9 (Dextroamphetamine);**
57-27-2 (Morphine)

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FILE 'HCAPLUS' ENTERED AT 15:35:23 ON 01 MAR 2003
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FILE COVERS 1907 - 1 Mar 2003 VOL 138 ISS 10
FILE LAST UPDATED: 28 Feb 2003 (20030228/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

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L169 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2003 ACS
AN **2002:521416** HCAPLUS
DN **137:57581**
TI Use of catecholamine reuptake inhibitors to enhance **memory**
IN **Epstein, Mel H.; Wiig, Kjesten A.**
PA **Sention, Inc., USA**
SO PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K
CC 1-11 (Pharmacology)
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002053104	A2	20020711	WO 2002-US34	20020102

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM,

HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS,
LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO,
RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ,
VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002161002 A1 20021031 US 2002-39229 20020102
PRAI US 2001-259374P P 20010102

AB The invention provides methods and reagents for enhancing **memory**, e.g., to increase **memory** function such as long-term **memory** and **recall** ability. The methodol. of the invention uses catecholamine reuptake inhibitors.

ST catecholamine reuptake inhibitor **memory** enhancement

IT AIDS (disease)
(AIDS dementia complex; catecholamine reuptake inhibitors to enhance **memory**)

IT Mental disorder
(AIDS dementia; catecholamine reuptake inhibitors to enhance **memory**)

IT Brain, disease
Prion diseases
(Creutzfeldt-Jakob, **memory** impairment assocd. with; catecholamine reuptake inhibitors to enhance **memory**)

IT Nervous system
(Huntington's chorea, **memory** impairment assocd. with; catecholamine reuptake inhibitors to enhance **memory**)

IT Mental disorder
(Pick's disease, **memory** impairment assocd. with; catecholamine reuptake inhibitors to enhance **memory**)

IT Nervous system
(adrenergic, adrenergic activators; catecholamine reuptake inhibitors to enhance **memory**)

IT Aging, animal
(age-assocd. **memory** impairment; catecholamine reuptake inhibitors to enhance **memory**)

IT Mental disorder
(attention deficit disorder; catecholamine reuptake inhibitors to enhance **memory**)

IT Mental disorder
(attention deficit hyperactivity disorder; catecholamine reuptake inhibitors to enhance **memory**)

IT Aneurysm
(brain, **memory** impairment assocd. with; catecholamine reuptake inhibitors to enhance **memory**)

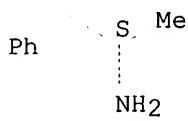
IT Alzheimer's disease
Amnesia
Anti-Alzheimer's agents
Anticonvulsants
Antidepressants
Antipsychotics
Anxiety
Anxiolytics
Cognition enhancers
Drug delivery systems
Drug interactions
Epilepsy
Human
Mental retardation
Nervous system agents
Schizophrenia
(catecholamine reuptake inhibitors to enhance **memory**)

IT Catecholamines, biological studies

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(catecholamine reuptake inhibitors to enhance **memory**)
IT Neurotrophic factors
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(catecholamine reuptake inhibitors to enhance **memory**)
IT Nervous system
(cholinergic, cholinergic activators; catecholamine reuptake inhibitors to enhance **memory**)
IT Mental disorder
(cognitive; catecholamine reuptake inhibitors to enhance **memory**)
IT Mental disorder
(dementia; catecholamine reuptake inhibitors to enhance **memory**)
IT Mental disorder
(depression; catecholamine reuptake inhibitors to enhance **memory**)
IT **Cognition**
Learning
Memory, biological
(disorder; catecholamine reuptake inhibitors to enhance **memory**)
IT Nervous system
(dopaminergic, dopaminergic activators; catecholamine reuptake inhibitors to enhance **memory**)
IT Nervous system
(**glutaminergic, glutaminergic** activators; catecholamine reuptake **inhibitors** to enhance **memory**)
IT Brain, disease
(injury; catecholamine reuptake inhibitors to enhance **memory**)
IT **Memory, biological**
(long-term; catecholamine reuptake inhibitors to enhance **memory**)
IT Toxicants
(**memory** impairment assocd. with exposure to; catecholamine reuptake inhibitors to enhance **memory**)
IT Parkinson's disease
(**memory** impairment assocd. with; catecholamine reuptake inhibitors to enhance **memory**)
IT Growth factors, animal
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(neuronal, and **neuronal survival factors**; catecholamine reuptake inhibitors to enhance **memory**)
IT Nerve
(noradrenergic; catecholamine reuptake inhibitors to enhance **memory**)
IT Drug delivery systems
(oral; catecholamine reuptake inhibitors to enhance **memory**)
IT Synapse
(presynapse; catecholamine reuptake inhibitors to enhance **memory**)
IT Amines, biological studies
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(secondary, tricyclic agents; catecholamine reuptake inhibitors to enhance **memory**)
IT Brain, disease
(stroke; catecholamine reuptake inhibitors to enhance **memory**)
IT Amines, biological studies
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL

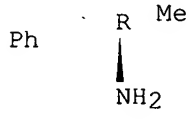
(Biological study); USES (Uses)
(tertiary, tricyclic agents; catecholamine reuptake inhibitors to enhance **memory**)
IT Drug delivery systems
(transdermal; catecholamine reuptake inhibitors to enhance **memory**)
IT Head
(trauma, **memory** impairment assocd. with; catecholamine reuptake inhibitors to enhance **memory**)
IT Biological transport
(uptake; catecholamine reuptake inhibitors to enhance **memory**)
IT Drugs
(veterinary; catecholamine reuptake inhibitors to enhance **memory**)
IT 51-41-2, Norepinephrine
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(catecholamine reuptake inhibitors to enhance **memory**)
IT 50-47-5, Desipramine 50-48-6, Amitriptyline 50-49-7, Imipramine **51-64-9**, S-(+)-**Amphetamine** 72-69-5, Nortriptyline 113-45-1, Methylphenidate **156-34-3**, R-(-)-**Amphetamine** **300-62-9**, **Amphetamine** 303-49-1, Clomipramine 438-60-8, Protriptyline 739-71-9, Trimipramine 1668-19-5, Doxepin 10262-69-8, Maprotiline 14028-44-5, Amoxapine 22232-71-9, Mazindol 24526-64-5, Nomifensine 53179-07-0, Nisoxetine 71620-89-8, Reboxetine 83366-66-9, Nefazodone 92623-85-3, Milnacipran 93413-69-5, Venlafaxine 106650-56-0, Sibutramine 116539-59-4, Duloxetine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(catecholamine reuptake inhibitors to enhance **memory**)
IT **141436-78-4**, **Protein kinase C** **142008-29-5**, **Protein kinase** A
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(pathway, stimulator; catecholamine reuptake inhibitors to enhance **memory**)
IT **51-64-9**, S-(+)-**Amphetamine** **156-34-3**, R-(-)-**Amphetamine** **300-62-9**, **Amphetamine**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(catecholamine reuptake inhibitors to enhance **memory**)
RN 51-64-9 HCAPLUS
CN Benzeneethanamine, .alpha.-methyl-, (.alpha.S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).

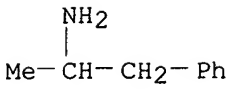


RN 156-34-3 HCAPLUS
CN Benzeneethanamine, .alpha.-methyl-, (.alpha.R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



RN 300-62-9 HCAPLUS
CN Benzeneethanamine, .alpha.-methyl- (9CI) (CA INDEX NAME)



IT **141436-78-4, Protein kinase C**
142008-29-5, Protein kinase A
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(pathway, stimulator; catecholamine reuptake inhibitors to enhance **memory**)
RN 141436-78-4 HCAPLUS
CN Kinase (phosphorylating), protein, C (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 142008-29-5 HCAPLUS
CN Kinase (phosphorylating), protein, A (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L169 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2003 ACS
AN **2002:391513** HCAPLUS
DN **136:380122**
TI Methods and **compositions** for regulating **memory** consolidation
IN **Epstein, Mel H.; Wiig, Kjesten A.**
PA **Sention, Inc., USA**
SO PCT Int. Appl., 130 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-00
CC 1-11 (Pharmacology)
Section cross-reference(s): 63
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002039998	A2	20020523	WO 2001-US45793	20011031
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2002039464	A5	20020527	AU 2002-39464	20011031
	US 2002115725	A1	20020822	US 2001-3740	20011031
PRAI	US 2000-245323P	P	20001101		
	WO 2001-US45793	W	20011031		

OS MARPAT 136:380122
AB The present invention makes available methods and reagents for enhancing and/or restoring long-term **memory** function and performance, e.g., to improve long-term **memory** (LTM) and **recall** ability in animal subjects.
ST **memory** consolidation enhancer
IT AIDS (disease)
(AIDS dementia complex; methods and **compns.** for enhancing **memory** consolidation)
IT Mental disorder
(AIDS dementia; methods and **compns.** for enhancing

memory consolidation)
IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**CREB** (cAMP-responsive element-binding), pathways; methods and **compns**. for enhancing **memory** consolidation)
IT Brain, disease
Prion diseases
(Creutzfeldt-Jakob; methods and **compns**. for enhancing **memory** consolidation)
IT Nervous system
(Huntington's chorea; methods and **compns**. for enhancing **memory** consolidation)
IT Mental disorder
(Pick's disease; methods and **compns**. for enhancing **memory** consolidation)
IT Brain, disease
(aneurysm; methods and **compns**. for enhancing **memory** consolidation)
IT Mental disorder
(attention deficit disorder; methods and **compns**. for enhancing **memory** consolidation)
IT Mental disorder
(attention deficit hyperactivity disorder; methods and **compns** . for enhancing **memory** consolidation)
IT Drug delivery systems
(carriers; methods and **compns**. for enhancing **memory** consolidation)
IT Aneurysm
(cerebral; methods and **compns**. for enhancing **memory** consolidation)
IT Mental disorder
(dementia; methods and **compns**. for enhancing **memory** consolidation)
IT **Learning**
(disorder; methods and **compns**. for enhancing **memory** consolidation)
IT Behavior
(inhibitory avoidance; methods and **compns**. for enhancing **memory** consolidation)
IT Brain, disease
(injury; methods and **compns**. for enhancing **memory** consolidation)
IT **Memory, biological**
(long-term; methods and **compns**. for enhancing **memory** consolidation)
IT **Adrenoceptor agonists**
Alzheimer's disease
Amnesia
Anti-Alzheimer's agents
Anticonvulsants
Antidepressants
Antiparkinsonian agents
Antipsychotics
Anxiolytics
Cholinergic agonists
Cognition enhancers
Dopamine agonists
Epilepsy
Human
Learning
Mammalia
Memory, biological
Mental retardation

Nervous system stimulants
Parkinson's disease
Permeation enhancers
Schizophrenia
(methods and **compns.** for enhancing **memory** consolidation)

IT Neurotrophic factors
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods and **compns.** for enhancing **memory** consolidation)

IT **Adrenoceptor agonists**
(noradrenergic; methods and **compns.** for enhancing **memory** consolidation)

IT Drug delivery systems
(oral; methods and **compns.** for enhancing **memory** consolidation)

IT **Cannabinoids**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(pathways; methods and **compns.** for enhancing **memory** consolidation)

IT Drug delivery systems
(prodrugs; methods and **compns.** for enhancing **memory** consolidation)

IT Brain, disease
(stroke; methods and **compns.** for enhancing **memory** consolidation)

IT Drug delivery systems
(transdermal, controlled-release, patches; methods and **compns** . for enhancing **memory** consolidation)

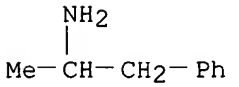
IT Head
(trauma; methods and **compns.** for enhancing **memory** consolidation)

IT 113-45-1, Methylphenidate **300-62-9D, Amphetamine,** derivs. **537-46-2 9061-61-4,** Nerve growth factor **33817-09-3**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods and **compns.** for enhancing **memory** consolidation)

IT **56-12-2, Gaba,** biological studies **487-79-6, Kainic acid 6384-92-5, Nmda 50812-31-2,** Cyclic nucleotide **phosphodiesterase 77521-29-0, Ampa 141436-78-4, Protein kinase c 142008-29-5, Protein kinase** a
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(pathways; methods and **compns.** for enhancing **memory** consolidation)

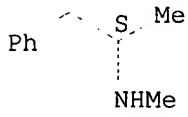
IT **300-62-9D, Amphetamine,** derivs. **537-46-2 9061-61-4,** Nerve growth factor **33817-09-3**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods and **compns.** for enhancing **memory** consolidation)

RN 300-62-9 HCAPLUS
CN Benzeneethanamine, .alpha.-methyl- (9CI) (CA INDEX NAME)



RN 537-46-2 HCAPLUS
CN Benzeneethanamine, N,.alpha.-dimethyl-, (.alpha.S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).

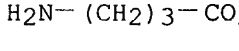


RN 9061-61-4 HCAPLUS
CN Nerve growth factor (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 33817-09-3 HCAPLUS
CN Benzeneethanamine, N,.alpha.-dimethyl-, (.alpha.R)- (9CI) (CA INDEX NAME)

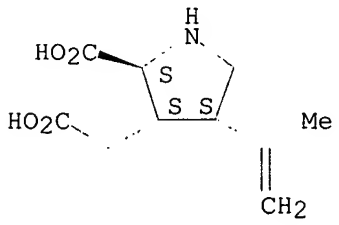
Absolute stereochemistry. Rotation (-).

IT **56-12-2, Gaba**, biological studies **487-79-6, Kainic acid 6384-92-5, Nmda 50812-31-2**, Cyclic nucleotide **phosphodiesterase 77521-29-0, Ampa 141436-78-4, Protein kinase c 142008-29-5, Protein kinase** a
RL: BSU (Biological study, unclassified); BIOL (Biological study) (pathways; methods and **compns**. for enhancing **memory** consolidation)
RN 56-12-2 HCAPLUS
CN Butanoic acid, 4-amino- (9CI) (CA INDEX NAME)



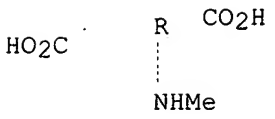
RN 487-79-6 HCAPLUS
CN 3-Pyrrolidineacetic acid, 2-carboxy-4-(1-methylethenyl)-, (2S,3S,4S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



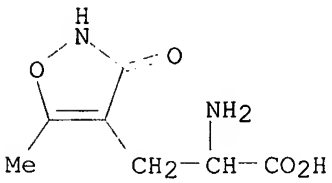
RN 6384-92-5 HCAPLUS
CN D-Aspartic acid, N-methyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 50812-31-2 HCAPLUS
CN Phosphodiesterase, cyclic nucleotide (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 77521-29-0 HCAPLUS
CN 4-Isoxazolepropanoic acid, .alpha.-amino-2,3-dihydro-5-methyl-3-oxo- (9CI) (CA INDEX NAME)



RN 141436-78-4 HCAPLUS
CN Kinase (phosphorylating), protein, C (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 142008-29-5 HCAPLUS
CN Kinase (phosphorylating), protein, A (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L169 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2003 ACS
AN 2002:171690 HCAPLUS
DN 136:210588
TI Use of methylphenidate compounds to enhance **memory**
IN **Wiig, Kjesten A.; Epstein, Mel H.**
PA **Sention, Inc, USA**
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-4458
ICS A61K031-445; A61K031-453; A61K009-70; A61P025-28
CC 1-11 (Pharmacology)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002017920	A2	20020307	WO 2001-US26829	20010828
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2001086861	A5	20020313	AU 2001-86861	20010828
PRAI	US 2000-228525P	P	20000828		
	US 2000-235971P	P	20000928		

US 2000-248278P P 20001114
WO 2001-US26829 W 20010828
OS MARPAT 136:210588
AB Methods and methylphenidate compds. are provided for facilitating LTP, e.g., to increase **memory** function such as long-term **memory** and **recall** ability.
ST methylphenidate compd **memory** enhancement; long term **memory recall** methylphenidate compd
IT **Cognition enhancers**
Stereoisomers
(methylphenidate compds. for **memory** enhancement)
IT Drug delivery systems
(prodrugs; methylphenidate compds. for **memory** enhancement)
IT Drug delivery systems
(transdermal; methylphenidate compds. for **memory** enhancement)
IT 113-45-1, Methylphenidate 113-45-1D, Methylphenidate, derivs. and prodrugs 20748-11-2 20748-12-3
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methylphenidate compds. for **memory** enhancement)

L169 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2003 ACS
AN 2002:171689 HCAPLUS
DN 136:210587
TI Use of threo-methylphenidate compounds to enhance **memory**
IN **Wiig, Kjesten A.; Epstein, Mel H.**
PA **Sention, Inc., USA**
SO PCT Int. Appl., 80 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-4458
ICS A61K031-45; A61K031-445; A61K031-453; A61K009-70; A61P025-28
CC 1-11 (Pharmacology)
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002017919	A2	20020307	WO 2001-US26774	20010828
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2001085325	A5	20020313	AU 2001-85325	20010828

PRAI US 2000-228478P P 20000828
US 2000-235972P P 20000928
WO 2001-US26774 W 20010828
OS MARPAT 136:210587
AB Methods and methylphenidate compds. are provided for facilitating **memory**, e.g., to increase **memory** function such as long-term **memory** and **recall** ability.
ST methylphenidate compd isomer **memory** enhancement
IT **Memory, biological**
(long-term; methylphenidate compds. to enhance **memory**)
IT **Cognition enhancers**
Drug delivery systems
Stereoisomers
(methylphenidate compds. to enhance **memory**)
IT Drug delivery systems

(prodrugs; methylphenidate compds. to enhance **memory**)
IT Drug delivery systems
(transdermal; methylphenidate compds. to enhance **memory**)
IT 113-45-1D, Methylphenidate, derivs. and prodrugs 40431-62-7 40431-62-7D, derivs. and prodrugs 40431-63-8 40431-63-8D, derivs. and prodrugs
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(methylphenidate compds. to enhance **memory**)
IT 113-45-1, Methylphenidate 40431-64-9 40572-71-2
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methylphenidate compds. to enhance **memory**)

L169 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2003 ACS
AN **2000:861482** HCAPLUS
DN **134:32977**
TI Methods and **compositions** for the treatment of neuroleptic and related disorders using sertindole derivatives
IN Jerussi, Thomas P.
PA Sepracor Inc., USA
SO PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-00
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1, 28
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000072837	A2	20001207	WO 2000-US14984	20000531
	WO 2000072837	A3	20010517		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6489341	B1	20021203	US 2000-580492	20000530
PRAI	US 1999-137447P	P	19990602		
	US 2000-580492	A	20000530		

AB The invention relates to novel methods using, and pharmaceutical **compns**. and dosage forms comprising, sertindole derivs. Sertindole derivs. include, but are not limited to, nor-sertindole, 5-oxo-sertindole, dehydro-sertindole, and dehydro-nor-sertindole. The methods of the invention are directed to the treatment and prevention of neuroleptic and related disorders such as, but are not limited to, psychotic disorders, depression, anxiety, substance addiction, **memory** impairment and pain. For example, capsules were prepd. contg. a sertindole deriv. 50.0 mg, lactose 48.5 mg, TiO2 0.5 mg, and Mg stearate 1.0 mg.
ST sertindole deriv prepn delivery system antipsychotic; anxiolytic sertindole deriv prepn delivery system; analgesic sertindole deriv prepn delivery system; antidepressant sertindole deriv delivery system; drug withdrawal sertindole deriv delivery system
IT 5-HT receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(5-HT2A, binding to; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Dopamine receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(D2, binding to; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Dopamine receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(D4, binding to; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT **Nervous system stimulants**
Psychotomimetics
(addiction and withdrawal; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT **Opioids**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(addiction and withdrawal; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Mental disorder
(affective; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT **Cholinergic agonists**
(analgesics; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Tachykinin receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(antagonists; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Heart, disease
(arrhythmia; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Drug delivery systems
(buccal; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Development, mammalian postnatal
(child; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Mental disorder
(cognitive, age-related; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Cardiovascular system
(disease; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT **Cognition**
(disorder, age-related; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT **Memory, biological**
(disorder; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Aging, animal
(elderly; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Heart, disease
(failure; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Mental disorder
(hysteria, psychosis; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Mental disorder
(manic bipolar disorder; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT Drug delivery systems
(mucosal; prepn. and **compns**. of sertindole derivs. for

treatment of neuroleptic and related disorders)
IT Nerve, disease
(neuropathy, pain; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Anti-inflammatory agents
(nonsteroidal; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Drug delivery systems
(oral; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Drug delivery systems
(parenterals; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT **5-HT agonists**
Adrenoceptor agonists
Alcoholism
Amnesia
Analgesics
Antiarrhythmics
Antidepressants
Antihypertensives
Antipsychotics
Antipyretics
Anxiolytics
Cognition enhancers
Drug dependence
Drug withdrawal
Hypertension
Obesity
Schizophrenia
Tobacco smoke
(prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Mental disorder
(psychosis, Cheyne-Stokes; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Arteriosclerosis
Menopause
Mental disorder
(psychosis; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Drug delivery systems
(sublingual; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Drug delivery systems
(topical; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Drug delivery systems
(transdermal; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT **Antidepressants**
(tricyclic; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT Adrenoceptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(.alpha.1, binding to; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT **Adrenoceptor antagonists**
(.alpha.1-; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)
IT 50-36-2, Cocaine 54-11-5, Nicotine 58-25-3, Chlordiazepoxide
64-17-5, Ethanol, biological studies 67-52-7D, 2,4,6(1H,3H,5H)-

Pyrimidinetrione, derivs. 72-44-6, Methaqualone 77-21-4, Glutethimide 113-18-8, Ethchlorvynol 125-64-4, Methyprylon **300-62-9D, Amphetamine**, derivs. 439-14-5, Diazepam 604-75-1, Oxazepam 846-50-4, Temazepam 28981-97-7, Alprazolam
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study) (addiction and withdrawal; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT 9002-17-9, Xanthine oxidase
RL: BSU (Biological study, unclassified); BIOL (Biological study) (inhibitors; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT 138900-27-3P
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); PROC (Process); RACT (Reactant or reagent); USES (Uses) (prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT 106516-07-8P 106516-24-9DP, Sertindole, derivs. 168274-35-9P 173294-84-3P
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses) (prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT 106516-24-9, Sertindole
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT 50-47-5, Desipramine 50-48-6 50-49-7, Imipramine 50-78-2, Aspirin 53-86-1, Indomethacin 60-99-1, Methotrimeprazine 72-69-5, Nortriptyline 103-90-2, Acetaminophen 315-30-0, Allopurinol 361-37-5, Methysergide 22071-15-4, Ketoprofen 54910-89-3, Fluoxetine 61869-08-7, Paroxetine 74103-06-3, Ketorolac 79617-96-2, Sertraline 85650-52-8, Mirtazapine 93413-69-5, Venlafaxine 116539-59-4, Duloxetine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); MOA (Modifier or additive use); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

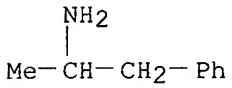
IT 540-49-8, 1,2-Dibromoethylene 1943-83-5, 2-Chloroethylisocyanate 41979-39-9, 4-Piperidone hydrochloride 180911-99-3
RL: RCT (Reactant); RACT (Reactant or reagent) (prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT 138900-22-8P, 1-(4-Fluorophenyl)-5-chlorindole 168274-49-5P 170232-37-8P 311330-26-4P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent) (prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT 50-67-9, Serotonin, biological studies 51-41-2, Norepinephrine
RL: BSU (Biological study, unclassified); BIOL (Biological study) (reuptake inhibitors; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

IT **300-62-9D, Amphetamine**, derivs.
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study) (addiction and withdrawal; prepn. and **compns**. of sertindole derivs. for treatment of neuroleptic and related disorders)

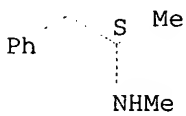
RN 300-62-9 HCAPLUS
CN Benzeneethanamine, .alpha.-methyl- (9CI) (CA INDEX NAME)



L169 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2003 ACS
AN **1996:447247** HCAPLUS
DN **125:104998**
TI Inhibition of cerebral **protein kinase C** in vitro by cocaine and **methamphetamine**
AU Morishita, Shigeru; Shimosato, Kazuaki; Saito, Taiichi
CS Department Psychiatry, Kawasaki Medical School, Kurashiki, 701-01, Japan
SO Kawasaki Medical Journal (1995), 21(1-2-3-4), 25-29
CODEN: KAMJDW; ISSN: 0385-0234
PB Kawasaki Medical School
DT Journal
LA English
CC 1-11 (Pharmacology)
Section cross-reference(s): 7
AB **Protein kinase C**, which participates in cellular responses to various stimuli such as hormones, neurotransmitters and growth factors, is essential for cell proliferation and differentiation. Activation of the enzyme has been suggested to be important in neurotransmitter release, learning and **memory**, long-term potentiation, and cocaine-induced motor activity. Our previous study showed that monoamine uptake inhibitors imipramine and desipramine inhibited **protein kinase C** activity in a crude ext. from the rat cerebral cortex. The present study examd. the effect of cocaine and **methamphetamine** on activity of the sol. **protein kinase C** in a crude ext. of the rat cerebral cortex. Cocaine and **methamphetamine** were found to inhibit **protein kinase C** in the sol. fraction at higher concns. It is, therefore, conceivable that the neural action of cocaine and **methamphetamine** may, at least in part, be assocd. with their inhibitory effect on **protein kinase C**.
ST **protein kinase C** inhibition cocaine **methamphetamine**; cerebral cortex **protein kinase** cocaine **methamphetamine**
IT **Nervous system agents**
(inhibition of cerebral **protein kinase C** in vitro by cocaine and **methamphetamine**)
IT Brain
(cerebral cortex, inhibition of cerebral **protein kinase C** in vitro by cocaine and **methamphetamine**)
IT 50-36-2, Cocaine **537-46-2, Methamphetamine**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibition of cerebral **protein kinase C** in vitro by cocaine and **methamphetamine**)
IT **141436-78-4, Protein kinase C**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of cerebral **protein kinase C** in vitro by cocaine and **methamphetamine**)
IT **537-46-2, Methamphetamine**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibition of cerebral **protein kinase C** in vitro by cocaine and **methamphetamine**)
RN 537-46-2 HCAPLUS
CN Benzeneethanamine, N,.alpha.-dimethyl-, (.alpha.S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



IT **141436-78-4, Protein kinase C**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of cerebral **protein kinase C** in vitro by cocaine and **methamphetamine**)
RN 141436-78-4 HCAPLUS
CN Kinase (phosphorylating), protein, C (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

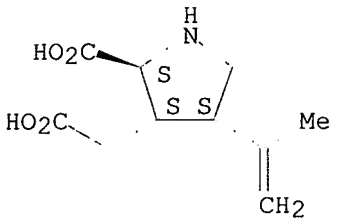
L169 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:341603 HCAPLUS
DN 122:123826
TI The role of angiotensin II in the regulation of blood flow to choroid plexuses and cerebrospinal fluid formation in the rat
AU Chodobski, Adam; Szmydynger-Chodobska, Joanna; **Epstein, Mel H.**; Johanson, Conrad E.
CS Department of Clinical Neurosciences, Brown University, Providence, RI, 02903, USA
SO Journal of Cerebral Blood Flow and Metabolism (1995), 15(1), 143-51
CODEN: JCBMDN; ISSN: 0271-678X
DT Journal
LA English
CC 2-10 (Mammalian Hormones)
AB The effect of peripherally administered angiotensin II (AII) on blood flow to choroid plexuses was examd. in pentobarbital-anesthetized rats. The indicator fractionation method with 123I- or 125I-N-isopropyl-p-**iodoamphetamine** as the marker was employed to measure blood flow. Basal blood flow to choroid plexus of the lateral cerebral ventricle (LVCP) (3.19 mL g-1 min-1) was lower than that to choroid plexuses of the 3rd (3VCP) and 4th (4VCP) ventricles (3.90 and 3.95 mL g-1 min-1, resp.). The effect of AII on choroidal blood flow varied depending on peptide dose and anatomical location of the choroidal tissue. AII infused i.v. at rates of 30 and 50 ng kg-1 min-1 decreased blood flow to both LVCP and 4VCP by 12-20%. Both lower (10 ng kg-1 min-1) and higher (100 and 300 ng kg-1 min-1) AII doses did not alter blood flow to LVCP and 4VCP. Blood flow to the 3VCP was not affected by any dose of the peptide used. In comparison, blood flow to cerebral cortex increased by 33% during i.v. AII infusion at a rate of 300 ng kg-1 min-1. The choroidal blood flow-lowering effect of moderate AII doses was abolished by both AT1 (losartan) and AT2 (PD 123319) receptor subtype antagonists (3 mg kg-1 i.v.). To det. whether the hemodynamic changes obsd. in choroid plexuses with moderate AII doses influence CSF formation, the ventriculocisternal perfusion was performed in rats (under the exptl. conditions described) with Blue Dextran 2000 as the indicator. CSF prodn. was not altered during i.v. infusion of AII at a rate of 30 ng kg-1 min-1. It is suggested that CSF formation is maintained in pathophysiol. situations

accompanied by increased plasma AII levels, which implicates a potential role for AII in regulating ion and water balance in the CNS.
ST angiotensin circulation choroid plexus cerebrospinal fluid
IT Cerebrospinal fluid
Circulation
(angiotensin II role in regulation of blood flow to choroid plexuses and cerebrospinal fluid formation)
IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(angiotensin II AT1, angiotensin II role in regulation of blood flow to choroid plexuses and cerebrospinal fluid formation)
IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(angiotensin II AT2, angiotensin II role in regulation of blood flow to choroid plexuses and cerebrospinal fluid formation)
IT Nervous system
(central, angiotensin II role in regulation of blood flow to choroid plexuses and cerebrospinal fluid formation)
IT Brain
(cerebral cortex, angiotensin II role in regulation of blood flow to choroid plexuses and cerebrospinal fluid formation)
IT Meninges
(choroid plexus, angiotensin II role in regulation of blood flow to choroid plexuses and cerebrospinal fluid formation)
IT 11128-99-7, Angiotensin-II
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(angiotensin II role in regulation of blood flow to choroid plexuses and cerebrospinal fluid formation)

L169 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2003 ACS
AN **1979:162674** HCAPLUS
DN **90:162674**
TI Avoidance, operant and locomotor behavior in rats with neostriatal injections of **kainic acid**
AU Sanberg, Paul R.; Pisa, Michele; Fibiger, Hans C.
CS Dep. Psychiatry, Univ. British Columbia, Vancouver, BC, Can.
SO Pharmacology, Biochemistry and Behavior (1979), 10(1), 137-44
CODEN: PBBHAU; ISSN: 0091-3057
DT Journal
LA English
CC 3-5 (Biochemical Interactions)
AB Compared with saline injected controls, rats with bilateral injections of **kainic acid** (KA) [**487-79-6**] in the dorsal neostriatum had increased locomotor response to d-**amphetamine**, increased resistance to extinction, and impaired acquisition and retention of passive avoidance. The KA injection resulted in loss of local neurons in the dorsal neostriatum, with no appreciable damage either to dopaminergic terminals or to extrinsic myelinated axons. Although loss of hippocampal neurons was occasionally obsd., the behavioral results could not be wholly attributed to hippocampal damage, since rats with no demonstrable extrastriatal lesions were not less impaired than those with hippocampal damage. An altered arousal reaction to stressful situations might account for the learning and **memory** impairments of the KA neostriatal rats.
ST **kainate** brain behavior
IT **Learning**
Memory, biological
(**kainate** effect on, brain damage in relation to)
IT Behavior
(locomotor, **kainate** effect on, brain damage in relation to)

IT Brain, toxic chemical and physical damage
(neostriatum, **kainate** toxicity to, behavior in relation)
IT **487-79-6**
RL: PRP (Properties)
(behavior response to, brain damage in relation to)
IT **487-79-6**
RL: PRP (Properties)
(behavior response to, brain damage in relation to)
RN 487-79-6 HCAPLUS
CN 3-Pyrrolidineacetic acid, 2-carboxy-4-(1-methylethenyl)-, (2S,3S,4S)-
(9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



=> sel hit rn
E1 THROUGH E13 ASSIGNED

=> fil reg
FILE 'REGISTRY' ENTERED AT 15:35:45 ON 01 MAR 2003
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provided by InfoChem.

STRUCTURE FILE UPDATES: 27 FEB 2003 HIGHEST RN 496010-47-0
DICTIONARY FILE UPDATES: 27 FEB 2003 HIGHEST RN 496010-47-0

TSCA INFORMATION NOW CURRENT THROUGH MAY 20, 2002

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. See HELP
PROPERTIES for more information. See STNote 27, Searching Properties
in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

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1 141436-78-4/BI
(141436-78-4/RN)
1 300-62-9/BI
(300-62-9/RN)
1 142008-29-5/BI
(142008-29-5/RN)
1 487-79-6/BI
(487-79-6/RN)

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             1 537-46-2/BI
               (537-46-2/RN)
             1 156-34-3/BI
               (156-34-3/RN)
             1 33817-09-3/BI
               (33817-09-3/RN)
             1 50812-31-2/BI
               (50812-31-2/RN)
             1 51-64-9/BI
               (51-64-9/RN)
             1 56-12-2/BI
               (56-12-2/RN)
             1 6384-92-5/BI
               (6384-92-5/RN)
             1 77521-29-0/BI
               (77521-29-0/RN)
             1 9061-61-4/BI
               (9061-61-4/RN)
L170        13 (141436-78-4/BI OR 300-62-9/BI OR 142008-29-5/BI OR 487-79-6/BI
               OR 537-46-2/BI OR 156-34-3/BI OR 33817-09-3/BI OR 50812-31-2/BI
               OR 51-64-9/BI OR 56-12-2/BI OR 6384-92-5/BI OR 77521-29-0/BI OR
               9061-61-4/BI)

=> d ide can tot

L170 ANSWER 1 OF 13  REGISTRY  COPYRIGHT 2003 ACS
RN   142008-29-5  REGISTRY
CN   Kinase (phosphorylating), protein, A (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   CAMP-dependent protein kinase
CN   CAMP-dependent protein kinase A
CN   Cyclic AMP-dependent protein kinase
CN   Cyclic AMP-dependent protein kinase A
CN   Heart muscle kinase
CN   Protein kinase A
CN   Protein kinase HMK
CN   Protein kinase Ukc1
CN   Protein kinase X
MF   Unspecified
CI   MAN
SR   CA
LC   STN Files:   ADISNEWS, AGRICOLA, BIOBUSINESS, BIOSIS, CA, CAPLUS, CEN,
       CHEMCATS, CIN, PROMT, TOXCENTER, USPAT2, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
            7946 REFERENCES IN FILE CA (1962 TO DATE)
              39 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
            7974 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE   1:  138:135829

REFERENCE   2:  138:135210

REFERENCE   3:  138:134435

REFERENCE   4:  138:134430

REFERENCE   5:  138:134415

REFERENCE   6:  138:134274

REFERENCE   7:  138:134248
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REFERENCE 8: 138:134230

REFERENCE 9: 138:134229

REFERENCE 10: 138:134228

L170 ANSWER 2 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **141436-78-4** REGISTRY
CN Kinase (phosphorylating), protein, C (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Calcium-dependent protein kinase C
CN Calcium/phospholipid-dependent protein kinase
CN Calcium/phospholipid-dependent protein kinase C
CN Conventional protein kinase C
CN Phosphatidylserine-sensitive calcium-dependent protein kinase
CN Protein kinase C
CN Protein kinase C.nu.
CN Protein kinase C3
CN Protein kinase PKC1
CN Type II protein kinase C
MF Unspecified
CI MAN
PCT Manual registration
SR CA
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CEN, CHEMCATS, CIN, CSCHEM, EMBASE, IPA, PROMT, TOXCENTER, USPAT2, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
21577 REFERENCES IN FILE CA (1962 TO DATE)
65 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
21628 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:135829

REFERENCE 2: 138:135564

REFERENCE 3: 138:134768

REFERENCE 4: 138:134486

REFERENCE 5: 138:134411

REFERENCE 6: 138:134400

REFERENCE 7: 138:134234

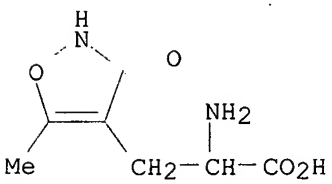
REFERENCE 8: 138:134230

REFERENCE 9: 138:134229

REFERENCE 10: 138:134001

L170 ANSWER 3 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **77521-29-0** REGISTRY
CN 4-Isoxazolepropanoic acid, .alpha.-amino-2,3-dihydro-5-methyl-3-oxo- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN (.+-.)-.alpha.-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid
CN (R,S)-.alpha.-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid
CN (RS)-.alpha.-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid
CN .alpha.-Amino-2,3-dihydro-5-methyl-3-oxoisoxazole-4-propionic acid
CN .alpha.-Amino-3-hydroxy-5-methyl-4-isoxazolepropionate

CN .alpha.-Amino-3-hydroxy-5-methylisoxazole-4-propionic acid
CN .gamma.-Amino-3-hydroxy-5-methylisoxazole-4-propionic acid
CN AMPA
CN AMPA (pharmaceutical)
CN D,L-.alpha.-Amino-3-hydroxy-5-methylisoxazole-4-propionic acid
CN dl-.alpha.-Amino-3-hydroxy-5-methylisoxazole-4-propionic acid
FS 3D CONCORD
DR 126632-03-9, 133481-32-0, 139261-99-7, 139559-02-7, 74341-63-2, 78729-80-3, 79697-77-1, 85506-19-0, 86495-63-8, 83354-19-2, 81323-87-7, 92614-50-1, 110592-37-5
MF C7 H10 N2 O4
CI COM
LC STN Files: BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CEN, CHEMCATS, CIN, CSCHEM, DDFU, DRUGU, EMBASE, MEDLINE, MRCK*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1112 REFERENCES IN FILE CA (1962 TO DATE)
9 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1114 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:135090

REFERENCE 2: 138:131461

REFERENCE 3: 138:103350

REFERENCE 4: 138:101195

REFERENCE 5: 138:101081

REFERENCE 6: 138:83736

REFERENCE 7: 138:83702

REFERENCE 8: 138:66947

REFERENCE 9: 138:66941

REFERENCE 10: 138:66939

L170 ANSWER 4 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **50812-31-2** REGISTRY
CN Phosphodiesterase, cyclic nucleotide (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Cyclic nucleotide phosphodiesterase
CN Cyclic nucleotide phosphohydrolase
MF Unspecified

CI MAN
LC STN Files: AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, EMBASE, PROMT, TOXCENTER, USPAT2, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
279 REFERENCES IN FILE CA (1962 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
280 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:67585

REFERENCE 2: 138:52349

REFERENCE 3: 137:365771

REFERENCE 4: 137:217245

REFERENCE 5: 137:83613

REFERENCE 6: 137:75227

REFERENCE 7: 136:380122

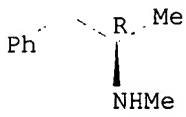
REFERENCE 8: 136:274002

REFERENCE 9: 136:194311

REFERENCE 10: 136:178015

L170 ANSWER 5 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **33817-09-3** REGISTRY
CN Benzeneethanamine, N,.alpha.-dimethyl-, (.alpha.R)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Benzeneethanamine, N,.alpha.-dimethyl-, (R)-
CN Phenethylamine, N,.alpha.-dimethyl-, (-)- (8CI)
OTHER NAMES:
CN (-)-Deoxyephedrine
CN (-)-Methamphetamine
CN (-)-N-Methylamphetamine
CN (R)-(-)-Deoxyephedrine
CN (R)-(-)-Methamphetamine
CN (R)-Deoxyephedrine
CN (R)-Methylamphetamine
CN (R)-N-Methylamphetamine
CN 2R-(-)-Methamphetamine
CN D-Methamphetamine
CN l-(-)-Methamphetamine
CN l-Methamphetamine
CN l-Methylamphetamine
CN Levmetamfetamine
CN R(-)-N-Methylamphetamine
CN Vicks Inhaler
FS STEREOSEARCH
DR 13897-80-8, 45952-93-0
MF C10 H15 N
CI COM
LC STN Files: ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, CA, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMLIST, CSCHEM, DDFU, DRUGU, IFICDB, IFIPAT, IFIUDB, PHARMASEARCH, PROMT, RTECS*, SPECINFO, TOXCENTER, USAN, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry. Rotation (-).



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

263 REFERENCES IN FILE CA (1962 TO DATE)
263 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:130563

REFERENCE 2: 138:51053

REFERENCE 3: 138:51040

REFERENCE 4: 138:19491

REFERENCE 5: 138:1269

REFERENCE 6: 137:364547

REFERENCE 7: 137:362116

REFERENCE 8: 137:227827

REFERENCE 9: 137:211249

REFERENCE 10: 137:210786

L170 ANSWER 6 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **9061-61-4** REGISTRY
CN Nerve growth factor (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Nerve growth hormone
CN NGF
MF Unspecified
CI PMS, COM, MAN
PCT Manual registration
LC STN Files: ADISINSIGHT, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CBNB, CEN, CHEMCATS, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, PHAR, PROMT, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
9100 REFERENCES IN FILE CA (1962 TO DATE)
125 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
9109 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:142438

REFERENCE 2: 138:134544

REFERENCE 3: 138:134401

REFERENCE 4: 138:134358

REFERENCE 5: 138:131524

REFERENCE 6: 138:131002

REFERENCE 7: 138:130773

REFERENCE 8: 138:122038

REFERENCE 9: 138:120924

REFERENCE 10: 138:120421

L170 ANSWER 7 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **6384-92-5** REGISTRY
CN D-Aspartic acid, N-methyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Aspartic acid, N-methyl-, D- (8CI)
OTHER NAMES:
CN 3: PN: US20030004099 SEQID: 13 claimed sequence
CN N-Methyl-D-aspartic acid
CN NMDA
FS STEREOSEARCH
MF C5 H9 N O4
CI COM
LC STN Files: AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, EMBASE, IFICDB, IFIUDB, IPA, MEDLINE, MRCK*, NIOSHTIC, PROMT, RTECS*, TOXCENTER, USPAT2, USPATFULL
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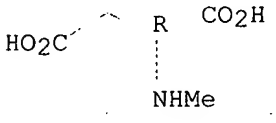
Absolute stereochemistry.



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9 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
6045 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:135074

REFERENCE 2: 138:134990

REFERENCE 3: 138:134987

REFERENCE 4: 138:131522

REFERENCE 5: 138:131479

REFERENCE 6: 138:131461

REFERENCE 7: 138:131455

REFERENCE 8: 138:131454

REFERENCE 9: 138:131451

REFERENCE 10: 138:130934

L170 ANSWER 8 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **537-46-2** REGISTRY
CN Benzeneethanamine, N,.alpha.-dimethyl-, (.alpha.S)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Benzeneethanamine, N,.alpha.-dimethyl-, (S)-
CN Phenethylamine, N,.alpha.-dimethyl-, (S)-(+)- (8CI)
OTHER NAMES:
CN (+)-(S)-Deoxyephedrine
CN (+)-2-(N-Methylamino)-1-phenylpropane
CN (+)-Methamphetamine
CN (+)-Methylamphetamine
CN (+)-N,.alpha.-Dimethyl-.beta.-phenylethylamine
CN (+)-N-Methylamphetamine
CN (S)-(+)-Deoxyephedrine
CN (S)-(+)-Methamphetamine
CN (S)-Methamphetamine
CN (S)-Methylamphetamine
CN 2S-(+)-Methamphetamine
CN d-(S)-Methamphetamine
CN d-Deoxyephedrine
CN d-Desoxyephedrine
CN d-Methamphetamine
CN d-Methylamphetamine
CN d-N,.alpha.-Dimethylphenethylamine
CN d-N-Methylamphetamine
CN d-Phenylisopropylmethylamine
CN L-Methamphetamine
CN Metamfetamine
CN Metamphetamine
CN Methamphetamine
CN Methyl-.beta.-phenylisopropylamine
CN Methylamphetamine
CN N-Methyl-1-phenyl-2-propanamine
CN N-Methylamphetamine
CN Norodin
FS STEREOSEARCH
DR 139-47-9, 1690-86-4, 14611-50-8, 45952-89-4
MF C10 H15 N
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGU, EMBASE, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, NIOSHTIC, PIRA, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL
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Other Sources: EINECS**, WHO
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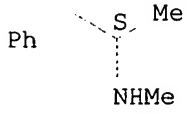
Absolute stereochemistry. Rotation (+).



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3398 REFERENCES IN FILE CA (1962 TO DATE)
79 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

3416 REFERENCES IN FILE CAPLUS (1962 TO DATE)
19 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 138:132330

REFERENCE 2: 138:132316

REFERENCE 3: 138:132315

REFERENCE 4: 138:130989

REFERENCE 5: 138:130792

REFERENCE 6: 138:130563

REFERENCE 7: 138:130454

REFERENCE 8: 138:130453

REFERENCE 9: 138:130452

REFERENCE 10: 138:122647

L170 ANSWER 9 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **487-79-6** REGISTRY
CN 3-Pyrrolidineacetic acid, 2-carboxy-4-(1-methylethenyl)-, (2S,3S,4S)-(9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 3-Pyrrolidineacetic acid, 2-carboxy-4-(1-methylethenyl)-, [2S-(2.alpha.,3.beta.,4.beta.)]-
CN 3-Pyrrolidineacetic acid, 2-carboxy-4-isopropenyl- (6CI, 7CI, 8CI)
OTHER NAMES:
CN (-)-.alpha.-Kainic acid
CN (-)-Kainic acid
CN (2S,3S,4S)-2-Carboxy-4-isopropenylpyrrolidine-3-acetic acid
CN .alpha.-Kainic acid
CN Digenic acid
CN Digenin
CN Helminal
CN Kainic acid
CN L-.alpha.-Kainic acid
FS STEREOSEARCH
DR 4071-38-9, 46398-96-3
MF C10 H15 N O4
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, HODOC*, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PROMT, RTECS*, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
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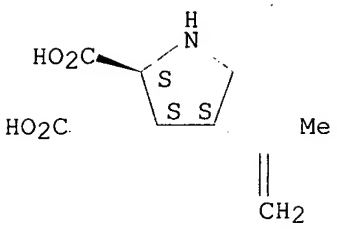
Other Sources: WHO

Absolute stereochemistry. Rotation (-).



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

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4177 REFERENCES IN FILE CAPLUS (1962 TO DATE)
26 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 138:135090

REFERENCE 2: 138:135076

REFERENCE 3: 138:135049

REFERENCE 4: 138:135011

REFERENCE 5: 138:134958

REFERENCE 6: 138:131461

REFERENCE 7: 138:118775

REFERENCE 8: 138:103350

REFERENCE 9: 138:101195

REFERENCE 10: 138:87826

L170 ANSWER 10 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **300-62-9** REGISTRY
CN Benzeneethanamine, .alpha.-methyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Benzeneethanamine, .alpha.-methyl-, (.+-.)-
CN Phenethylamine, .alpha.-methyl-, (.+-.)- (8CI)
OTHER NAMES:
CN (.+-.)-.alpha.-Methylphenethylamine
CN (.+-.)-.alpha.-Methylphenylethylamine
CN (.+-.)-.beta.-Phenylisopropylamine
CN (.+-.)-1-Phenyl-2-aminopropane
CN (.+-.)-Desoxynorephedrine
CN (.+-.)-Phenylisopropylamine
CN .alpha.-Methyl-.beta.-phenylethylamine
CN .alpha.-Methylbenzeneethanamine
CN .alpha.-Methylphenethylamine
CN .alpha.-Methylphenylethylamine
CN .beta.-Aminopropylbenzene
CN .beta.-Phenylisopropylamine
CN 1-Benzylethylamine
CN 1-Methyl-2-phenylethylamine
CN 1-Phenyl-2-aminopropane
CN 1-Phenyl-2-propanamine
CN 1-Phenyl-2-propylamine

CN 2-Amino-1-phenylpropane
CN 3-Phenyl-2-propylamine
CN Actedron
CN Adderall
CN Adderall XR
CN Adipan
CN Allodene
CN Amfetamine
CN Amphetamine
CN Anorexine
CN Benzebar
CN Benzedrine
CN Benzolone
CN Desoxynorephedrine
CN dl-.alpha.-Methylphenethylamine
CN Elastonon
CN Fenopromin
CN Finam
CN Isoamyne
CN Isomyn
CN Mecodrin
CN Norephedrane
CN Novydrine
CN Obesin
CN Obesine
CN Oktedrin
CN Ortedrine
CN Percomon
CN Phenamine
CN Phenedrine
CN Profamina
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
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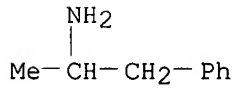
DR 60-15-1, 17108-96-2, 96332-84-2
MF C9 H13 N
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, DDFU, DETHERM*, DIOGENES, DRUGU, EMBASE, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PHARMASEARCH, PIRA, PROMT, RTECS*, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

6227 REFERENCES IN FILE CA (1962 TO DATE)
461 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
6241 REFERENCES IN FILE CAPLUS (1962 TO DATE)
5 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 138:135000

REFERENCE 2: 138:133514

REFERENCE 3: 138:132330

REFERENCE 4: 138:132316

REFERENCE 5: 138:132315

REFERENCE 6: 138:130982

REFERENCE 7: 138:130966

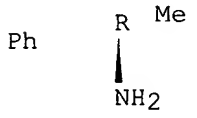
REFERENCE 8: 138:126950

REFERENCE 9: 138:122647

REFERENCE 10: 138:118594

L170 ANSWER 11 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **156-34-3** REGISTRY
CN Benzeneethanamine, .alpha.-methyl-, (.alpha.R)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Benzeneethanamine, .alpha.-methyl-, (R)-
CN Phenethylamine, .alpha.-methyl-, (-)- (8CI)
OTHER NAMES:
CN (-)-(R)-Amphetamine
CN (-)-Amphetamine
CN (-)-Phenaminum
CN (-)-Phenylisopropylamine
CN (2R)-(-)-Amphetamine
CN (R)-(-)-Amphetamine
CN (R)-(-)-Amphetamine
CN (R)-.alpha.-Methylphenethylamine
CN (R)-1-Methyl-2-phenylethylamine
CN (R)-1-Phenyl-2-aminopropane
CN (R)-1-Phenyl-2-propylamine
CN (R)-Amphetamine
CN (R)-Amphetamine
CN L-(-)-Amphetamine
CN l-(-)-Amphetamine
CN l-.alpha.-Methylphenethylamine
CN l-Amphetamine
CN L-Amphetamine
CN Levamfetamine
CN Levoamphetamine
FS STEREOSEARCH
MF C9 H13 N
CI COM
LC STN Files: ADISNEWS, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, GMELIN*, IFICDB, IFIPAT, IFIUDB, IPA, MRCK*, RTECS*, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry. Rotation (-).



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

626 REFERENCES IN FILE CA (1962 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
627 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:122647

REFERENCE 2: 138:51053

REFERENCE 3: 138:51040

REFERENCE 4: 138:33353

REFERENCE 5: 138:1269

REFERENCE 6: 137:370075

REFERENCE 7: 137:364547

REFERENCE 8: 137:227827

REFERENCE 9: 137:210786

REFERENCE 10: 137:179318

L170 ANSWER 12 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **56-12-2** REGISTRY
CN Butanoic acid, 4-amino- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Butyric acid, 4-amino- (7CI, 8CI)
OTHER NAMES:
CN .gamma.-Aminobutanoic acid
CN .gamma.-Aminobutryic acid
CN .gamma.-Aminobutyric acid
CN .omega.-Aminobutyric acid
CN 3-Carboxypropylamine
CN 4-Aminobutanoic acid
CN 4-Aminobutyric acid
CN Aminalon
CN GABA
CN Gaballon
CN Gamarex
CN Gammalon
CN Gammalone
CN Gammar
CN Gammasol
CN Mielogen
CN Mielomade
CN Piperidic acid
CN Piperidinic acid
FS 3D CONCORD
DR 3131-86-0
MF C4 H9 N O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DETHERM*, DRUGU, EMBASE, GMELIN*, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, ULIDAT, USAN, USPAT2, USPATFULL, VETU

(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

$H_2N-(CH_2)_3-CO_2H$

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

24347 REFERENCES IN FILE CA (1962 TO DATE)
426 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
24368 REFERENCES IN FILE CAPLUS (1962 TO DATE)
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REFERENCE 1: 138:137552

REFERENCE 2: 138:136472

REFERENCE 3: 138:136258

REFERENCE 4: 138:136104

REFERENCE 5: 138:135091

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REFERENCE 7: 138:135049

REFERENCE 8: 138:134973

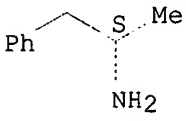
REFERENCE 9: 138:134133

REFERENCE 10: 138:134076

L170 ANSWER 13 OF 13 REGISTRY COPYRIGHT 2003 ACS
RN **51-64-9** REGISTRY
CN Benzeneethanamine, .alpha.-methyl-, (.alpha.S)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Benzeneethanamine, .alpha.-methyl-, (S)-
CN Phenethylamine, .alpha.-methyl-, (+)- (8CI)
OTHER NAMES:
CN (+)-(S)-Amphetamine
CN (+)-.alpha.-Methylphenethylamine
CN (+)-Amphetamine
CN (+)-Phenaminum
CN (2S)-(+)-Amphetamine
CN (S)-(+)-.beta.-Phenylisopropylamine
CN (S)-(+)-Amphetamine
CN (S)-.alpha.-Methylphenethylamine
CN (S)-1-Phenyl-2-aminopropane
CN (S)-1-Phenyl-2-propylamine
CN (S)-Amphetamine
CN D-(+)-Amphetamine
CN d-(S)-Amphetamine
CN d-.alpha.-Methylphenethylamine
CN d-Amphetamine
CN D-Amphetamine
CN Dexadrine
CN Dexamfetamine
CN Dexamphetamine
CN Dextroamphetamine

CN NSC 73713
FS STEREOSEARCH
MF C9 H13 N
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGU, EMBASE, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PROMT, RTECS*, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry. Rotation (+).



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

4136 REFERENCES IN FILE CA (1962 TO DATE)
16 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
4140 REFERENCES IN FILE CAPLUS (1962 TO DATE)
18 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 138:131001

REFERENCE 2: 138:130994

REFERENCE 3: 138:130990

REFERENCE 4: 138:130932

REFERENCE 5: 138:122647

REFERENCE 6: 138:120337

REFERENCE 7: 138:119226

REFERENCE 8: 138:117593

REFERENCE 9: 138:100951

REFERENCE 10: 138:100811

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FILE LAST UPDATED: 28 Feb 2003 (20030228/ED)

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L182 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2003 ACS
AN **2002:520340** HCAPLUS
DN **137:211249**
TI Phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning**
AU Reed, Tracy M.; Repaske, David R.; Snyder, Gretchen L.; Greengard, Paul; Vorhees, Charles V.
CS Division of Developmental Biology, Children's Hospital Research Foundation, Cincinnati, OH, 45229, USA
SO Journal of Neuroscience (2002), 22(12), 5188-5197
CODEN: JNRSDS; ISSN: 0270-6474
PB Society for Neuroscience
DT Journal
LA English
CC 2-8 (Mammalian Hormones)
AB Using homologous recombination, we generated mice lacking phosphodiesterase-mediated (PDE1B) cyclic nucleotide-hydrolyzing activity. PDE1B-/- mice showed exaggerated hyperactivity after acute D-methamphetamine administration. Striatal slices from PDE1B-/- mice exhibited increased levels of phospho-Thr34 DARPP-32 and phospho-Ser845 GluR1 after dopamine D1 receptor agonist or forskolin stimulation. PDE1B-/- and PDE1B+/- mice demonstrated Morris maze spatial-**learning** deficits. These results indicate that enhancement of cyclic nucleotide signaling by inactivation of PDE1B-mediated cyclic nucleotide hydrolysis plays a significant role in dopaminergic function through the DARPP-32 and related transduction pathways.
ST phosphodiesterase 1B locomotor DARPP32 phosphorylation dopamine **learning**
IT Phosphoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(DARPP-32 (dopamine-cAMP-regulated phosphoprotein, 32,000-mol.-wt.); phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)
IT Dopamine receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(D1; phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)
IT Glutamate receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GluR1 subunit; phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)
IT Brain
(corpus striatum; phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in

response to dopamine agonists and display impaired spatial **learning** in mice)

IT Behavior
(locomotor; phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)

IT Signal transduction, biological
(phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)

IT Phosphorylation, biological
(protein; phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)

IT **Learning**
(spatial; phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)

IT 9040-59-9, Calcium/calmodulin-dependent phosphodiesterase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(isoenzyme 1B; phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)

IT **33817-09-3**, D-Methamphetamine
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)

RE.CNT 74 THERE ARE 74 CITED REFERENCES AVAILABLE FOR THIS RECORD

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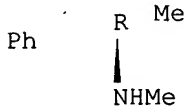
IT **33817-09-3**, D-Methamphetamine
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(phosphodiesterase 1B knock-out mice exhibit exaggerated locomotor hyperactivity and DARPP-32 phosphorylation in response to dopamine agonists and display impaired spatial **learning** in mice)

RN 33817-09-3 HCAPLUS
CN Benzeneethanamine, N,.alpha.-dimethyl-, (.alpha.R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L182 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2003 ACS
AN **2000:404793** HCAPLUS
DN **133:129796**
TI Adult **learning** deficits after neonatal exposure to D-methamphetamine: selective effects on spatial navigation and **memory**
AU Vorhees, Charles V.; Inman-Wood, Sandra L.; Morford, LaRonda L.; Broening, Harry W.; Fukumura, Masao; Moran, Mary S.
CS Division of Developmental Biology, Children's Hospital Research Foundation and Department of Pediatrics, University of Cincinnati, Cincinnati, OH, 45229-3039, USA
SO Journal of Neuroscience (2000), 20(12), 4732-4739
CODEN: JNRSDS; ISSN: 0270-6474
PB Society for Neuroscience
DT Journal
LA English
CC 1-11 (Pharmacology)
AB The effects of neonatal D-methamphetamine (MA) treatment on cued and spatial **learning** and **memory** were investigated. MA was administered to neonatal rats on postnatal days 11-20. All groups received four s.c. injections per day. Group MA40-4 received 40 mg.cntdot.kg-1.cntdot.d-1 of MA in four divided doses (10 mg/kg per injection). Group MA40-2 received 40 mg.cntdot.kg-1.cntdot.d-1 of MA in two divided (20 mg/kg/injection) and saline for the other two injections per day. Controls received saline for four injections per day. As adults, both MA groups showed no differences in swimming ability in a straight swimming channel. The MA40-4 group showed no differences in cued **learning**, but was impaired in hidden platform **learning** in the Morris water maze on acquisition. They also showed reduced **memory** performance on probe trials. Similar trends were seen on reversal **learning** and reversal probe trials. Reduced platform-size **learning** trials caused spatial **learning** impairments to re-emerge in the MA40-4 group. The MA40-2 group showed no differences in straight channel swimming, but was slower at finding the visible platform during cued **learning**. They were also impaired during acquisition and **memory** trials in the Morris hidden platform maze. They showed a similar trend on reversal **learning** and **memory** trials, but were not different during reduced platform-size **learning** trials. When the MA40-2 group's performance on hidden platform **learning** and **memory** trials was adjusted for cued trial performance, the spatial **learning** deficits remained. Deficits of spatial **learning** and **memory** are a selective effect of neonatal methamphetamine treatment irresp. of other **learning** and performance variables.
ST neonate methamphetamine **learning** deficit **memory**
IT **Learning**
Memory, biological
(adult **learning** deficits after neonatal exposure to D-methamphetamine and selective effects on spatial navigation and **memory**)
IT **Learning**
(spatial; adult **learning** deficits after neonatal exposure to D-methamphetamine and selective effects on spatial navigation and **memory**)
IT **33817-09-3**, D-Methamphetamine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(adult **learning** deficits after neonatal exposure to D-methamphetamine and selective effects on spatial navigation and **memory**)
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
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IT **33817-09-3**, D-Methamphetamine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(adult **learning** deficits after neonatal exposure to D-methamphetamine and selective effects on spatial navigation and **memory**)

RN 33817-09-3 HCAPLUS

CN Benzeneethanamine, N,.alpha.-dimethyl-, (.alpha.R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).


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=> fil wpix
FILE 'WPIX' ENTERED AT 15:46:51 ON 01 MAR 2003
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FILE LAST UPDATED:          27 FEB 2003      <20030227/UP>
MOST RECENT DERWENT UPDATE:     200314        <200314/DW>
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    available in the /ABEX field. An additional search field
    /BIX is also provided which comprises both /BI and /ABEX <<<

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    PLEASE VISIT:
 http://www.stn-international.de/training_center/patents/stn_guide.pdf <<<

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    http://www.derwent.com/userguides/dwpi_guide.html <<<

=> d all abeq tech abex tot l185

L185 ANSWER 1 OF 4  WPIX (C) 2003 THOMSON DERWENT
AN   2002-599589 [64]   WPIX
DNC  C2002-169413
TI   Use of a formulation of a catecholamine reuptake inhibitor for enhancing
     long-term memory.
DC   B05
IN   EPSTEIN, M; WIIG, K A; EPSTEIN, M H
PA   (EPST-I) EPSTEIN M; (WIIG-I) WIIG K A; (SENT-N) SENTION INC
CYC  96
PI   WO 2002053104 A2 20020711 (200264)* EN   51p    A61K000-00      <--
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            NL OA PT SD SE SL SZ TR TZ UG ZM ZW
         W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
            DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ
            LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD
            SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
     US 2002161002 A1 20021031 (200274)              A61K031-551     <--
ADT  WO 2002053104 A2 WO 2002-US34 20020102; US 2002161002 A1
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Provisional US 2001-259374P 20010102, US 2002-39229 20020102

PRAI **US 2001-259374P 20010102; US 2002-39229 20020102**

IC ICM A61K000-00; A61K031-551
ICS A61K031-137

AB WO 200253104 A UPAB: 20021007
NOVELTY - Enhancing long term memory in an animal involves administering a formulation of a catecholamine reuptake inhibitor (A).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a medicament for enhancing memory in animal comprising a formulation;

(2) preparation of a formulation for enhancing memory consolidation involves preparing a pharmaceutical preparation comprising at least one (A);

(3) a kit comprising at least one (A) provided in a single oral dosage form or as a transdermal patch in association with instructions (written and/or pictorial) describing the use of the kit and optionally, warnings of possible side effects and drug-drug or drug-food interactions;

(4) a method for conduction of a pharmaceutical business involving:

(i) manufacturing the kit and marketing to healthcare providers the benefits of using the kit or medicament;

(ii) providing distribution network for selling the kit or medicament and providing instruction material to patients or physicians for using the kit or medicament;

(iii) determining dosage of (A), conducting therapeutic profiling of at least one formulations of (A) for efficacy and toxicity in animals and providing a distribution network for selling the formulation; and

(iv) licensing to a third party, the rights for further development and sale of the (A).

ACTIVITY - Nootropic; Antidepressant; Neuroleptic; Neuroprotective; Tranquilizer; Cerebroprotective; Anticonvulsant; Antiparkinsonian; Vulnerary.

MECHANISM OF ACTION - Catecholamine reuptake inhibitor.

USE - The catecholamine reuptake inhibitor is used for enhancing long-term memory functions in normal individual and in veterinary treatment of animal; and also for treatment of anxiety, depression, age-associated memory impairment, minimal cognitive impairment, amnesia, dementia, learning disabilities, memory impairment, memory impairment associated with toxicant exposure, brain injury, stroke, schizophrenia, epilepsy, mental retardation, Alzheimer's disease, age attention deficit disorder, attention deficit hyperactivity disorder, AIDS-related dementia, brain aneurysm, Parkinson's disease, head trauma, Huntington's disease, Pick's disease, Creutzfeldt-Jakob disease in animal or human (all claimed).

ADVANTAGE - The norepinephrine reuptake inhibitor inhibits presynaptic norepinephrine reuptake with Ki of at most 100 nM and has 10 times greater selectivity for blocking norepinephrine reuptake as compared to inhibition of dopamine and serotonin (5-HT). The norepinephrine reuptake inhibitor is 10 times more potent at blocking noradrenergic neurons as compared to serotonergic neurons.

Dwg.0/22

FS CPI

FA AB; GI; DCN

MC CPI: B04-H06D; B08-D03; B11-C04; B12-M02F; B14-D02; B14-J01A1; B14-J01A3; B14-J01A4; B14-J01B3; B14-J01B4; B14-J02C1; B14-J07; B14-N16; B14-N16B; B14-S12

TECH UPTX: 20021007
TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Components: (A) is norepinephrine reuptake inhibitor. Preferably it is a tert-amine tricyclics or secondary amine tricyclics.
Preferred Method: The animal is further dosed with a neuronal growth factor, a neuronal survival factor, a neuronal tropic factor, a

cholinergic activator, an adrenergic activator, a dopaminergic activator, a glutaminergic activator or an agent that stimulates the PKC or PKA pathways. (A) is provided in an amount assayed by a standardized performance test such as at least one of Cambridge Neuropsychological Test Automated Battery (CANTAB), Children's Memory Scale (CMS), Contextual Memory Test, Continuous Recognition Memory Test (CMRT), Denman Neuropsychology Memory Scale, Fuld Object Memory Evaluation (FOME), Graham-Kendall Memory for Designs Test, Guild Memory Test, Learning and Memory Battery (LAMB), Memory Assessment Clinic Self Rating Scale (MAC-S), Memory Assessment Scales (MAS), Randt Memory Test, Recognition Memory Test (RMT); Rivermead Behavioral Memory Test, Russell's Version of the Wechsler Memory Scale (RWMS), Test of Memory and Learning (TOMAL), Vermont Memory Scale (VMS), Wechsler Memory Scale or Wide Range Assessment of Memory and Learning (WRAML) (preferably Providence Recognition Memory Test).

ABEX

SPECIFIC COMPOUNDS - Amitriptyline (I), clomipramine, doxepin, imipramine, trimipramine, amoxapine, desipramine, maprotiline, nortriptyline, protriptyline, reboxetine, duloxetine, venlafaxine, milnacipran, mazindol, methylphenidate, nefazodone, nisoxetine, sibutramine and nomifensine are specifically claimed as (A).

ADMINISTRATION - The dosage of (A) is 0.0001 - 100 mg/kg/day. (A) can be administered orally, parenterally (including intravenously, intramuscularly, intraarterially, intrathecally, intracapsularly, intraorbitally, intracardiacly, intradermally, intraperitoneally, transtracheally, subcutaneously, subcuticulaly, intraarticulaly, subcapsularly, subarachnoidly, intraspinally and intrasternal injection and infusion), topically, nasally or rectally.

EXAMPLE - Rats were injected with 3 different doses of methylphenidate (50, 100 and 150 standard units/kg) 30 minutes prior to training on the inhibitory task (IA). It was observed that a dose of 50 standard units/kg improved retention of IA. An unpaired t-test demonstrated that this enhancement was statistically significant (p less than 0.03).

L185 ANSWER 2 OF 4 WPIX (C) 2003 THOMSON DERWENT
AN 2002-479430 [51] WPIX
DNC C2002-136333
TI Enhancing memory consolidation comprises administration of methylphenidate formulation.
DC B05
IN EPSTEIN, M H; WIIG, K A
PA (SENT-N) SENTION INC
CYC 95
PI WO 2002017920 A2 20020307 (200251)* EN 68p A61K031-4458 <--
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001086861 A 20020313 (200251) A61K031-4458 <--
ADT WO 2002017920 A2 **WO 2001-US26829 20010828**; AU 2001086861 A **AU 2001-86861 20010828**
FDT AU 2001086861 A Based on WO 200217920
PRAI **US 2000-248278P 20001114; US 2000-228525P 20000828 ; US 2000-235971P 20000928**
IC ICM A61K031-4458
ICS A61K009-70; A61K031-445; A61K031-453; A61P025-28
AB WO 200217920 A UPAB: 20020812
NOVELTY - Enhancement of memory consolidation involves administering a formulation of methylphenidate compound (I) or its derivative, salt, solvate, pro-drug, or metabolic derivative.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(A) a transdermal patch comprising (I) or its analog;

(B) a method for conducting a pharmaceutical business involving either:

(1) manufacturing the transdermal patch, and

(2) marketing to healthcare providers the benefits of using the transdermal patch to increase memory function; or

(3) providing a distribution network for selling the transdermal patch and

(4) providing instruction material to patients or physicians for using the patch to increase memory function; or

(5) determining an appropriate transdermal patch and dosage of (I) in the transdermal patch to increase memory function,

(6) conducting therapeutic profiling of the transdermal patch identified in step (5) for efficacy and toxicity in animals and

(7) providing a distribution network for selling the patch identified in step (6) as having the therapeutic profile; or

(8) carrying our step (5) and

(9) licensing to a third party the rights for further development and sale of the transdermal patch; and

(C) a kit comprising (I), in an association with instructions (written and/or pictorial) describing the use of the formulation for enhancing memory, and optionally warnings of possible side effect and drug-drug or drug-food interactions.

ACTIVITY - Anticonvulsant; Nootropic; Neuroleptic; Antiparkinsonian; Neuroprotective; Cardiant; Cerebroprotective; Tranquilizer; Anti-HIV; Antidepressant.

MECHANISM OF ACTION - None given.

USE - For enhancing memory consolidation in an animal (claimed); as a neuroprotective treatment) preventing or slowing degradation of long-term memory function and performance; for restoring long-term memory function and performance; for treating and preventing memory impairment e.g. due to toxicant exposure, brain injury, age-associated memory impairment, mild cognitive impairment, epilepsy, mental retardation in children, and dementia resulting from a disease, such as Parkinson's disease, Alzheimer's disease, AIDS, head trauma, Huntington's disease, Pick's disease, Creutzfeldt-Jakob disease, Anterior communicating artery syndrome, hypoxia, post cardiac surgery, Down's syndrome and stroke, learning disorder, schizophrenia, senile dementia, drugs, or anatomical lesions (dementia), attention deficit disorder (ADD), attention-deficit hyperactivity disorder (ADHD), AIDS-related dementia. The memory disorders are functional mechanism (anxiety, depression), physiological ageing (age-associated memory impairment, mild cognitive impairment, etc.).

ADVANTAGE - The formulation facilitates the increase memory function such as long-term memory and recall ability and enhances the memory consolidation. The preparation reduces side-effects of racemic methylphenidate. The side-effects are insomnia, palpitation, headache, dyskinesia, drowsiness, tachycardia, angina, cardiac arrhythmia, abdominal pain, hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiform with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura), anorexia, appetite suppression, irritability, attentional sticking, dizziness and dysphoria, increased aggression, and stunted growth.

Dwg.0/3

FS CPI

FA AB; GI; DCN

MC CPI: B07-H; B11-C09; B12-M02F; B14-J01A2; B14-J01A3; B14-J01A4; B14-J01B3; B14-J01B4; B14-J07; B14-K01; B14-N16

TECH UPTX: 20020812

TECHNOLOGY FOCUS - ORGANIC CHEMISTRY - Preferred Compound: (I) is of formula Q-V-U-V-R2 (Ia). The metabolite of (I) is of formula (Ib).

A = carbocylic, heterocyclic, or (hetero)aryl (preferably (hetero)aryl);

Q = a group of formula (i) or (ii);
U = bond, -C(=O)-, -C(=S)-, -P(=O)(OR8)-, -S(O2)- or -S(O)- (preferably -C(=O)- or - C(=S)-);
V = bond, or NR, O or S (preferably present, especially NH, S or O);
Y = NR4, O or S;
X = C, N, S, Se or O;
R = H, lower alkyl, lower alkenyl, (hetero)aryl, or (hetero)aralkyl;
R1 = aryl, 1-6C acyloxy, cyano, amido, amino, 1-6C acylamino, 1-6C alkylamino, sulfonic acid or T;
T = 1-6C alkyl, 1-6C alkoxy, 1-6C alkanoyl, hydroxyl, halo, carboxyl, nitro, or sulfhydryl;
R2 = H, 1-6C alkyl or 1-6C alkanoyl (preferably H or 1-6C alkyl);
R3 = T, H, or 2-6C alkanoxyl;
R3+R3 = oxo or double bond between two adjacent X atoms;
R4 = H, lower alkyl, acyl, amido, ester, aryl, aralkyl, heteroaryl, or heteroaralkyl (preferably H or lower alkyl);
R8 = not defined;
m = 0 - 1;
n = 0 - 7;
p = 3 - 6;
q = 0 - 16;
s = 0 - 2;
Ar = optionally substituted (hetero)aryl;
t = 1 -6;
R5 = absent, hydroxyl or O-glucuronide;
Z = -CH2- or -C(=O)-;
T' = H or -C(=O)-NH2;
G = carboxylic acid or its salt, carboxylic acid methyl ester, carboxylic acid ethyl ester, carboxylic acid O-glucuronide or acetylamino ethane sulfonic acid.
Preferred Formulation: The ratio of DL-erythro stereoisomer of (I) to DL-threo stereoisomer of (I) is 1:4 - 1:1. The formulation is substantially free of erythro stereoisomers.
Preferred Method: The method additionally involves a step of providing a sales group for marketing the preparation to healthcare providers.
Preferred Patch: The transdermal patch further comprises at least one penetration enhancer.

ABEX

ADMINISTRATION - The formulation is administered in a single dosage form or as a transdermal patch (claimed). The formulation is also administered orally, parenterally (including intravenously, intramuscularly, intraarterially, intrathecally, intracapsularly, intraorbitally, intracardiacally, intradermally, intraperitoneally, transtracheally, subcutaneously, subcuticularly, intraarticularly, or subcapsularly, intraspinally, through intrasternal injection, infusion or subarachnoid injection), enterally, topically, nasally, intravaginally, intracisternally, bucally, sublingually, rectally, or intracerebroventricularly in a dosage of 1 - 90 (preferably 5 - 70, especially 10 - 30)%. The dosage for intravenous, intracerebroventricular, and subcutaneous administration is 0.0001 - 100 mg/kg of the body weight/day.

EXAMPLE - Rats were injected with three different doses of methylphenidate thirty minutes prior to training on the inhibitory avoidance task. The dose of 5 mg/kg had no effect. The dose of 5 mg/kg was most effective when administered to the rats one hour prior to training. In order to determine whether the enhanced retention was long-lasting, the rats were received a second retention test one week after the first retention test. No additional training or drug was administered to the animals in the interim period. The results demonstrated that performance of the methylphenidate-injected rats was still significantly enhanced one week following the original training session (t (54)= 2.358, with p less than 0.0220).

L185 ANSWER 3 OF 4 WPIX (C) 2003 THOMSON DERWENT
AN 2002-479429 [51] WPIX
DNC C2002-136332
TI Pharmaceutical preparation useful for enhancing memory consolidation comprises threo-methylphenidate compound.
DC B05
IN EPSTEIN, M; WIIG, K A; EPSTEIN, M H
PA (SENT-N) SENTION INC; (EPST-I) EPSTEIN M; (WIIG-I) WIIG K A
CYC 95
PI WO 2002017919 A2 20020307 (200251)* EN 80p A61K031-4458 <--
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001085325 A 20020313 (200251) A61K031-4458 <--
US 2002103162 A1 20020801 (200253) A61K031-675
US 2002132793 A1 20020919 (200264) A61K031-675
ADT WO 2002017919 A2 **WO 2001-US26774 20010828**; AU 2001085325 A **AU 2001-85325 20010828**; US 2002103162 A1 **Provisional US 2000-228478P 20000828, Provisional US 2000-235972P 20000928** , US 2001-941238 20010828; US 2002132793 A1 **Provisional US 2000-228478P 20000828, Provisional US 2000-235972P 20000928** , CIP of US 2001-941238 20010828, US 2002-87232 20020228
FDT AU 2001085325 A Based on WO 200217919
PRAI **US 2000-235972P 20000928; US 2000-228478P 20000828** ; US 2001-941238 20010828; US 2002-87232 20020228
IC ICM A61K031-4458; A61K031-675
ICS A61K009-70; A61K031-38; A61K031-397; A61K031-40; A61K031-445; A61K031-45; A61K031-453; A61P025-28; G06F017-60
AB WO 200217919 A UPAB: 20021031
NOVELTY - A pharmaceutical preparation comprises a methylphenidate compound (I) or its salt, solvate, pro-drug, or metabolic derivative.

DETAILED DESCRIPTION - A pharmaceutical preparation comprises a methylphenidate compound (I) or its salt, solvate, pro-drug, or metabolic derivative. The formulation includes either

(i) L-threo (2S:2'S) stereoisomer and/or D-threo (2R:2'R) stereoisomer of (I) (at least 60 w/w.%) relative to erythro- isomers of (I); or

(ii) L-threo (2S:2'S) stereoisomer of (I) relative to D-threo (2R:2'R), and D-erythro (2R:2'S) and L-erythro (2S:2'R) isomers of (I) (at least 60 w/w.%).

INDEPENDENT CLAIMS are also included for:

(1) a method for conducting a pharmaceutical business involving manufacturing the preparation, and marketing to healthcare providers the benefits of using the preparation to increase memory function;

(2) a method for conducting a pharmaceutical business involving providing a distribution network for selling the preparation, and providing instruction material to patients or physicians for using the preparation to increase memory function;

(3) a method for conducting a pharmaceutical business involving

(4) a method for conducting a pharmaceutical business involving determining an appropriate preparation and dosage of (I) to increase memory function, conducting therapeutic profiling of preparations for efficacy and toxicity in animals and providing a distribution network for selling a preparation identified in step (2b) as having the therapeutic profile;

(5) a method for conducting a pharmaceutical business comprising determining an appropriate preparation and dosage of methylphenidate to be administered toi increase memory function and licensing, to a third party, the rights for further development and sale of the preparation;

(6) a kit comprising the preparation containing (I) (where the preparation includes L-threo (2S:2'S) stereoisomer and/or D-threo (2R:2'R) stereoisomer of (I) (at least 60 w/w.%) relative to erythro- isomers of (I)) and instructions written and/or pictorial, describing the use of the preparation for enhancing memory in a patient.

ACTIVITY - Anticonvulsant; Nootropic; Neuroleptic; Antiparkinsonian; Neuroprotective; Cardiant; Cerebroprotective; Tranquilizer; Anti-HIV; Antidepressant. Rats were injected with three different doses of methylphenidate thirty minutes prior to training on the inhibitory avoidance task. The dose of 5 mg/kg had no effect. The dose of 5 mg/kg was most effective when administered to the rats one hour prior to training. In order to determine whether the enhanced retention was long-lasting, the rats were received a second retention test one week after the first retention test. No additional training or drug was administered to the animals in the interim period. The results demonstrated that performance of the methylphenidate-injected rats was still significantly enhanced one week following the original training session (t (54)= 2.358, with p less than 0.0220).

MECHANISM OF ACTION - None given.

USE - For enhancing memory consolidation in an animal (claimed); as a neuroprotective treatment preventing or slowing degradation of long-term memory function and performance; for restoring long-term memory function and performance; for treating and/or preventing memory impairment e.g. due to toxicant exposure, brain injury, age-associated memory impairment, mild cognitive impairment, epilepsy, mental retardation in children, and dementia resulting from a disease, such as Parkinson's disease, Alzheimer's disease, AIDS, head trauma, Huntington's disease, Pick's disease, Creutzfeldt-Jakob, Anterior communicating artery syndrome, hypoxia, post cardiac surgery, Down's syndrome and stroke, learning disorder, schizophrenia, senile dementia, drugs, or anatomical lesions (dementia), attention deficit disorder (ADD), attention-deficit hyperactivity disorder (ADHD), AIDS-related dementia. The memory disorders are functional mechanism (anxiety, depression), physiological ageing (age-associated memory impairment, mild cognitive impairment, etc).

ADVANTAGE - The preparation facilitates the memory e.g. to increase memory function such as long-term memory and recall ability and enhances the memory consolidation. The preparation reduces side-effects of racemic methylphenidate. The side-effects are insomnia, palpitation, headache, dyskinesia, drowsiness, tachycardia, angina, cardiac arrhythmia, abdominal pain, hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiform with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura), anorexia, appetite suppression, irritability, attentional sticking, dizziness and dysphoria, increased aggression, and stunted growth.

Dwg.0/9

FS CPI

FA AB; GI; DCN

MC CPI: B07-H; B14-A02B1; B14-F02D; B14-J01A; B14-J01B4; B14-J07; B14-N16

TECH UPTX: 20020812

TECHNOLOGY FOCUS - ORGANIC CHEMISTRY - Preferred Compound: (I) is of formula (Ia) or (Ib). The L-threo (2S:2'S) stereoisomer of (I) is of formula (Ic), (Id), (Ie), or (If).

A = carbocylic, heterocyclic, or (hetero)aryl (preferably (hetero)aryl);

U = bond, -C(=O)-, -C(=S)-, -P(=O)(OR8)-, -S(O2)- or -S(O)-(preferably -C(=O)- or - C(=S)-);

V = bond or NR, O or S (preferably NH, S or O);

Y = NR4, O or S;

X = C, N, S, Se or O;

R = H, lower alkyl, lower alkenyl, (hetero)aryl, or (hetero)aralkyl;

R1 = aryl, 1-6C acyloxy, cyano, amido, amino, 1-6C acylamino, 1-6C alkylamino, sulfonic acid or T;

T = 1-6C alkyl, 1-6C alkoxy, 1-6C alkanoyl, hydroxyl, halo, carboxyl, nitro, or sulfhydryl;

R2 = H, 1-6C alkyl or 1-6C alkanoyl (preferably H or 1-6C alkyl);
R3 = T, H, 2-6C or alkanoxy;
R3+R3 = oxo or double bond between two adjacent X atoms;
R4 = H, lower alkyl, acyl, amido, ester, aryl, aralkyl, heteroaryl, or heteroaralkyl (preferably H or lower alkyl);
R8 = not defined;
m = 0 - 1;
n = 0 - 7;
p = 3 - 6;
q = 0 - 16;
s = 0 - 2;
Ar = optionally substituted (hetero)aryl;
L = non-toxic organic or inorganic acid and/or quaternizing agent;
t = 1 - 6;
R5 = absent, hydroxyl or O-glucuronide;
Z = -CH2- or -C(=O)-;
T' = H or -C(=O)-NH2; and
G = carboxylic acid or its salt, carboxylic acid methyl ester, carboxylic acid ethyl ester, carboxylic acid O-glucuronide or acetylamino ethane sulfonic acid.
Preferred Method: The method additionally involves a step of providing a sales group for marketing the preparation to healthcare providers.

ABEX

ADMINISTRATION - The preparation is administered in a single dosage form or as a transdermal patch (claimed). The preparation is also administered orally, parenterally (including intravenously, intramuscularly, intraarterially, intrathecally, intracapsularly, intraorbitally, intracardiacally, intradermally, intraperitoneally, transtracheally, subcutaneously, subcuticularly, intraarticularly, or subcapsularly, intraspinally, or through intrasternal injection, and infusion or subarachnoid injection), enterally, topically, nasally, intravaginally, intracisternally, bucally, sublingually, rectally, or intracerebroventricularly in a dosage of 1 - 90 (preferably 5 - 70, especially 10 - 30)%. The dosage for intravenous, intracerebroventricular, and subcutaneous administration is 0.0001 - 100 mg/kg of the body weight/day.

L185 ANSWER 4 OF 4 WPIX (C) 2003 THOMSON DERWENT
AN 2002-454828 [48] WPIX
DNC C2002-129387
TI Use of amphetamine compound for enhancing long-term memory and for treatment of e.g. anxiety, depression, age-associated memory impairment, amnesia, dementia, learning difficulties and Parkinson's disease.
DC B05
IN EPSTEIN, M; WIIG, K A; EPSTEIN, M H
PA (EPST-I) EPSTEIN M; (WIIG-I) WIIG K A; (SENT-N) SENTION INC
CYC 95
PI WO 2002039998 A2 20020523 (200248)* EN 130p A61K031-00 <--
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
US 2002115725 A1 20020822 (200258) A61K031-137 <--
AU 2002039464 A 20020527 (200261) A61K031-00 <--
ADT WO 2002039998 A2 **WO 2001-US45793 20011031**; US 2002115725 A1 **Provisional US 2000-245323P 20001101, US 2001-3740 20011031**; AU 2002039464 A **AU 2002-39464 20011031**
FDT AU 2002039464 A Based on WO 200239998
PRAI **US 2000-245323P 20001101; US 2001-3740 20011031**
IC ICM A61K031-00; A61K031-137
AB WO 200239998 A UPAB: 20020730

NOVELTY - Pharmaceutical preparation comprises at least one amphetamine compound.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) A kit comprising the preparation; and

(2) Conducting a pharmaceutical business involving either:

(i) manufacturing the kit; and

(ii) marketing to healthcare providers the benefits of using the kit or preparation to enhance memory of treated patients;

(iii) providing a distribution network for selling the kit; and

(iv) providing instruction material to patients or physicians for using it or preparation to enhance memory of treated patients;

(v) determining an appropriate dosage of the amphetamine compound to enhance memory function in a class of patients;

(vi) conducting therapeutic profiling of at least one formulation of step (v) for efficacy and toxicity in animals; and

(vii) providing a distribution network for selling the formulation of step (vi); or

(viii) the step (v); and

(ix) licensing to a third party the rights for further development and sale of the amphetamine compound for enhancing memory.

ACTIVITY - Tranqulizer; Antidepressant; Nootropic; Antiparkinsonian; Vulnerary; Anticonvulsant; Cerebroprotective; Neuroleptic; Neuroprotective; Anti-HIV.

MECHANISM OF ACTION - None given.

USE - In the manufacture of a medicament for treatment of an animal (preferably mammal, particularly human) susceptible to or suffering from anxiety, depression, age-associated memory impairment, minimal cognitive impairment, amnesia, dementia, learning disabilities, memory impairment associated with toxicant exposure, brain injury, brain aneurysm, Parkinson's disease, head trauma, Huntington's disease, Pick's disease, Creutzfeldt-Jakob disease, stroke, schizophrenia, epilepsy, mental retardation, Alzheimer's disease, age, attention deficit disorder, attention deficit hyperactivity disorder, or AIDS-related dementia (all claimed).

ADVANTAGE - The preparation is formulated for sustained release of the amphetamine to enhance long-term memory in a patient but resulting in a concentration in the patient lower than its EC50 as a CNS stimulant. The preparation enhances long-term memory in a patient by statistically significant amount when assessed by a at least one of standardized performance test; Cambridge Neuropsychological Test Automated Battery (CANTAB); a Children's Memory Scale (CMS); a Contextual Memory Test; a Continuous Recognition Memory Test (CMRT); a Denman Neuropsychology Memory Scale; a Fuld Object; Memory Evaluation (FOME); a Graham-Kendall Memory for Designs Test; a Guild Memory Test; a Learning and Memory Battery (LAMB); a Memory Assessment Clinic Self Rating Scale (MAC-S); a Memory Assessment Scales (MAS); a Randt Memory Test; a Recognition Memory Test (RMT); a Rivermead Behavioral Memory Test; a Russell's Version of the Wechsler Memory Scale (RWMS); a Test of Memory and Learning (TOMAL); a Vermont Memory Scale (VMS); a Wechsler Memory Scale; and a Wide Range Assessment of Memory and Learning (WRAML).

Dwg.0/16

FS CPI

FA AB; GI; DCN

MC CPI: B04-H01; B06-H; B07-H; B10-A08; B10-A09B; B10-A10; B10-B04B; B12-M02F; B12-M10A; B14-J01; B14-J01A1; B14-J01A3; B14-J01A4; B14-J01B3; B14-J01B4; B14-J07; B14-N16

TECH UPTX: 20020730

TECHNOLOGY FOCUS - ORGANIC CHEMISTRY - Preferred Components: The amphetamine compound is of formulae (I), (II), (III) or its salts (preferably saccharate, sulfate or aspartate), solvates, metabolites or pro-drugs.

R1 = T' (preferably H or lower alkyl, particularly H);

T' = H, optionally substituted lower alkyl, lower alkenyl, lower alkynyl, aralkyl, aryl, heteroaralkyl, heteroaryl, cycloalkyl or cycloalkylalkyl;
R2 = T'' or optionally substituted lower alkyl (preferably H or lower alkyl, particularly H or methyl);
T'' = H, lower alkenyl, lower alkynyl, aralkyl, aryl, heteroaralkyl, heteroaryl, cycloalkyl or cycloalkylalkyl;
R3 = T''' or optionally substituted lower alkyl (preferably H or lower alkyl, especially H);
T''' = H, lower alkenyl, lower alkynyl, aralkyl, aryl, heteroaralkyl, heteroaryl, cycloalkyl or cycloalkylalkyl;
R4 = Q or sulfonate ester (preferably H, halo, trifluoromethyl, OH, amino, cyano, nitro or lower alkyl, particularly H);
Q = H, halo, OH, alkoxy, amino, alkylamino, sulfhydryl, alkylthio, cyano, nitro, ester, ketone, formyl, amido, acylamino, acyloxy, lower alkyl, lower alkenyl, amidino, sulfonyl, sulfoxido, sulfamoyl or sulfonamido;
L = non-toxic organic or inorganic acid;
R'4 = Q or ester (preferably H);
R'1 = T' (optionally substituted by halo, OH or alkoxy) (preferably H or lower alkyl, particularly H);
R'2 = T' or lower alkyl (H or lower alkyl, particularly H or methyl);
R'3 = T' or lower alkyl (preferably H or lower alkyl, particularly H); and
R5 = H or OH.
At least one (preferably at least two) of R1-R3 or R'1-R'3 is H.

TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Kit: The kit further comprises a neuronal growth factor, neuronal survival factor, neuronal trophic factor, cholinergic modulator, an adrenergic modulator, a nonadrenergic modulator, a dopaminergic modulator, a glutaminergic modulator, methylphenidate or an agent that stimulates the PKC, PKA, GABA, NMDA, cannabinoid, AMPA, kainate, phosphodiesterase (PDE), CREB or nootropic pathways. The kit comprises a single (preferably at least two species). The amphetamine compound is provided as at least 51 (preferably at least 75, more preferably at least 75, especially at least 95, particularly 99) mole % of the eutomer with respect to the distomer of that amphetamine compound.
Preferred Method: The method further includes providing a sales group for marketing the preparation to healthcare providers.

ABEX

ADMINISTRATION - The preparation is administered orally or in the form of transdermal patch which comprises at least one penetration enhancer (claimed). The preparation is administered enterally, nasally, rectally, vaginally, parenterally, topically (including buccally and sublingually), intravenously, intramuscularly, intraarterially, intrathecally, intracapsulalry, intraorbitally, intracardiacally, intradermally, intraperitonealy, transtracheally, subcutaneously, subcuticularly, intraarticulalry, subcapsulary, subarachnoid, intraspinally and by intrasternal injection and infusion.

EXAMPLE - Rats were injected with three different doses of S-(+)amphetamine, 30 minutes prior to training on inhibitory avoidance task. Results are not given.

=> d his

(FILE 'HOME' ENTERED AT 14:15:24 ON 01 MAR 2003)
SET COST OFF

FILE 'REGISTRY' ENTERED AT 14:15:44 ON 01 MAR 2003
E AMPHETAMINE/CN
L1 1 S E3
L2 193 S C9H13N/MF AND 46.150.18/RID AND 1/NR
L3 28 S L2 AND BENZENEETHANAMINE

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L4           18 S L3 AND ALPHA METHYL
L5            4 S L4 NOT (LABELED OR ION OR (D OR T)/ELS OR 11C# OR 13C# OR 14C
                E METHAMPHETAMINE/CN
L6            1 S E3
L7          331 S C10H15N/MF AND 46.150.18/RID AND 1/NR
L8           53 S L7 AND BENZENEETHANAMINE
L9            4 S L8 AND N ALPHA DIMETHYL
L10           3 S L9 NOT D/ELS
L11           3 S L5 NOT 13N
L12           6 S L1,L6,L10,L11
                SEL RN
L13         314 S E1-E6/CRN
L14          73 S L13 NOT ((MXS OR IDS)/CI OR COMPD)
L15          71 S L14 NOT CONJUGATE
L16          70 S L15 NOT B/ELS
L17          66 S L16 NOT (WITH OR CR/ELS)
L18          72 S L12,L17

     FILE 'MEDLINE' ENTERED AT 14:21:36 ON 01 MAR 2003
L19       16621 S L12
L20       16621 S L18
L21       25485 S ?AMPHETAMINE?
                E AMPHETAMINE/CT
                E E3+ALL
L22       12997 S E64+NT
L23       19400 S E64/CN,BI

     FILE 'REGISTRY' ENTERED AT 14:22:28 ON 01 MAR 2003
                SEL CHEM L12

     FILE 'MEDLINE' ENTERED AT 14:22:37 ON 01 MAR 2003
L24       24036 S E1-E168
L25        7415 S L24 NOT L19,L20
L26       25612 S L19,L20,L21,L22,L23,L24,L25
                E MEMORY/CT
                E E3+ALL
L27       34782 S E13+NT
                E MEMORY/CT
                E E11+ALL
L28       10224 S E10+NT
                E MEMOR/CT
L29       72316 S MEMORY
L30        7402 S AMNESI?
L31        9947 S AMNESTI?
L32         919 S KORSAKOF#
L33         573 S L26 AND L27-L32
                E NEURONAL GROWTH FACTOR/CT
L34          57 S NEURONAL GROWTH FACTOR
                E NERVE GROWTH FACTOR/CT
                E E3+ALL
L35       15723 S E61+NT OR E61/BI
L36        7368 S NGF
L37          29 S NEURONAL SURVIVAL FACTOR
                E NERVE SURVIVAL FACTOR
L38           1 S NERVE SURVIVAL FACTOR
L39          11 S NEURONAL TROPHIC FACTOR
L40           6 S CHOLINERGIC MODULATOR
                E CHOLINERGIC MODULATOR/CT
                E E6+ALL
                E E2+ALL
L41      105571 S E7+NT
                E ADRENERGIC MODULATOR/CT
L42           5 S E3/BI
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                E ADRENERGIC/CT
                E E4+ALL
L43        263468 S E7+NT
L44             0 S NONADRENERGIC MODULATOR
L45             0 S NON ADRENERGIC MODULATOR
L46          2064 S (NONADRENERGIC OR NON ADRENERGIC)(L)(MODULAT? OR AFFECT? OR I
L47             2 S DOPAMINERGIC MODULATOR
                E DOPAMINE/CT
L48        112544 S E6+NT
L49             0 S GLUTAMINERGIC MODULATOR
                E GLUTAMINERGIC/CT
                E GLUTAMINE/CT
L50          5922 S GLUTAMIN?(L)(MODULAT? OR AFFECT? OR INHIBIT? OR BLOCK? OR ANT
L51         15929 S PKC
L52         35495 S PROTEIN KINASE C
                E PROTEIN KINASE C/CT
L53         24868 S E3+NT
L54          8891 S PKA
L55         89671 S PROTEINKINASE OR PROTEIN KINASE
                E PROTEIN KINASE/CT
                E E48+ALL
L56        124921 S E7+NT
L57         33206 S GABA
                E GABA/CT
                E E8+ALL
L58         90623 S E7+NT
L59         25931 S GAMMA AMINOBUTYRIC ACID
L60           636 S GAMMA AMINO BUTYRIC ACID
L61         17516 S NMDA
                E NMDA/CT
                E E3+ALL
                E E2_ALL
                E NMDA/CT
                E E3+ALL
                E E2+ALL
L62          6084 S E23+NT
L63         19704 S N METHYLASPARTATE OR N METHYL (1W) (ASPARTATE OR ASPARTIC ACI
L64          3942 S CANNABINOID
                E CANNABINOID/CT
                E E4+ALL
L65          5458 S E5+NT
L66          5913 S AMPA
                E  AMPA/CT
                E E3+ALL
L67          1619 S E2
                E E2+ALL
L68          2054 S E14/BI
L69          4911 S KAINATE
                E KAINATE/CT
                E E3+ALL
                E E2+ALL
L70          5852 S E21+NT
L71          7581 S E21/BI
L72         22023 S PHOSPHODIESTERASE OR PDE
                E PHOSPHODIESTERASE/CT
                E E54+ALL
L73         34879 S E2+NT
L74          2945 S CREB
                E DNA-BINDING PROTEIN/CT
                E E4+ALL
L75          2430 S E9+NT
L76           995 S E13-E15,E18,E19/BI
L77            12 S NOOTROP?(L)PATHWAY
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                E HALLUCINOGEN/CT
L78         14597 S E8+NT
L79           468 S L33 AND L34-L78
L80           332 S L79 AND L19,L20
L81           406 S L79 AND L24
L82           468 S L79-L81
L83           405 S L82 AND PY<=2000
L84            38 S L83 AND L22(L)TU/CT
L85           215 S L83 AND L22(L)(AD OR PD OR PK)/CT
L86           138 S L83 AND L22/MAJ
L87           135 S L84,L85 AND L86
L88            40 S L87 NOT AB/FA
                E DRUG COMBINATION/CT
                E E6+ALL
L89         34925 S E4+NT
                E DRUG THERAPY, COMBINATION/CT
                E E3+ALL
L90         72196 S E4+NT
L91             5 S L89,L90 AND L83
                E AMITRIPTYLINE+ALL/CT
L92            50 S L87 AND (COADMIN? OR COMEDI? OR COPRESCRI? OR COTHERAP? OR CO
L93             3 S L88 AND L92
L94             8 S L91,L93
L95            44 S L92 NOT L94
                SEL DN AN 7 8 10 11 15-18 21 23 25 32 35 36 37 39 40
L96            17 S L95 AND E1-E51
L97            25 S L94,L96
L98         26516 S L27/MAJ OR L28/MAJ
                E RECALL/CT
                E E3+ALL
                E E2+ALL
L99           395 S E14+NT
L100        26681 S L98,L99
L101          162 S L19,L20 AND L99,L100
                E AMPHETAMINE+ALL/CT
L102        19400 S E64/BI,CN,CT
L103          173 S L98-L100 AND L102
L104          192 S L101,L103 AND PY<=2000
L105           57 S L104 NOT AB/FA
                SEL DN AN 4 11 21 28 32 34 35 50 57
L106            9 S L105 AND E1-E27
L107           33 S L97,L106
L108          123 S L104 NOT L105-L107
                SEL DN AN 94
L109            1 S E28-E30
L110           34 S L107,L109 AND L19-L109
L111           34 S L110 AND (MEMOR? OR RECAL? OR IMPAIR? OR AMNES? OR KORSAKOF?)

     FILE 'MEDLINE' ENTERED AT 15:07:12 ON 01 MAR 2003

     FILE 'HCAPLUS' ENTERED AT 15:07:23 ON 01 MAR 2003
L112        15733 S L12 OR L18
                E AMPHETAMINE/CT
                E E3+ALL
L113        22116 S ?AMPHETAMIN?
L114        23996 S L112,L113
                E MEMORY/CT
                E E3+ALL
L115        10497 S E1
                E E2+ALL
L116         7919 S E3,E1+NT
                E MEMMORY/CT
                E MEMORY/CT
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                E E4+ALL
                E E2+ALL
L117       10422 S E3+NT
                E ANMES/CT
                E AMNES/CT
L118        1397 S E4-E7
                E E4+ALL
L119        1397 S E5+NT
                E RECALL/CT
L120       89446 S MEMORY OR AMNES? OR RECALL
L121         419 S L114 AND L115-L120
L122      227827 S L34,L36-L40,L42,L44-L47,L49-L52,L54,L55,L57,L59-L61,L63,L64,L
L123         400 S L114 AND (REMEMBER? OR FORGET? OR MEMOR?)
L124          33 S L121,L123 AND L122

     FILE 'REGISTRY' ENTERED AT 15:14:27 ON 01 MAR 2003
L125           3 S PROTEIN KINASE C/CN
                E PKA/CN
                E GABA/CN
L126           1 S E3
                E NMDA/CN
L127           1 S E3
                E AMPA/CN
                E KAINIC ACID/CN
L128           1 S E3
L129        1237 S PHOSPHODIESTERASE
L130        1237 S L129 AND 1/NC
L131         168 S CREB

     FILE 'HCAPLUS' ENTERED AT 15:16:32 ON 01 MAR 2003
L132       83932 S L125,L126,L127,L128,L129,L131
L133          10 S L132 AND L121,L123
L134          33 S L124,L133
                E NEURONAL GROWTH FACTOR/CT
                E NERVE GROWTH FACTOR/CT
L135        9113 S E3
                E E3+ALL
L136         225 S E4
                E NEURONAL SURVIVAL FACTOR/CT
                E NERVE SURVIVAL FACTOR/CT
                E NERVE TROPHIC FACTOR/CT
                E NEURONAL TROPHIC FACTOR/CT
                E CHOLINERGIC /CT
                E E4+ALL
L137        2630 S E2+NT
                E CHOLINERGIC /CT
                E E10+ALL
L138        5153 S E6,E7,E5+NT
                E ADRNERGIC/CT
                E ADRENERGIC/CT
L139        5456 S E14+NT OR E23+NT
                E E14+ALL
                E E2+ALL
L140        7611 S E8,E9,E6+NT
                E ADRENERGIC/CT
                E E23+ALL
L141        3350 S E2
                E E2+ALL
L142       10814 S E7,E8,E5+NT
                E DOPAMINE/CT
L143        2438 S E5+NT OR E9+NT
                E E5+ALL
L144        2917 S E7,E6+NT
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                E DOPAMINE/CT
                E E9+ALL
L145        1939 S E6,E5+NT
                E GLUTAMINERG/CT
                E GLUTAMINE/CT
                E CANNABINOID/CT
L146        5835 S E10+NT
                E E10+ALL
                E NOOTROP/CT
                E E5+ALL
L147        1577 S E2+NT
                E E2+ALL
L148         390 S E6
L149      158353 S E3+NT
                E E3+ALL
                E MENTAL ACTIVITY/CT
L150       27724 S E3+NT
                E E3+ALL
L151         987 S L114 AND L150
L152        1087 S L121,L151,L123 AND L115-L120,L151
L153         320 S L152 AND L122,L135-L149
L154          99 S L153 AND MEMOR?
L155           6 S L154,L134 AND COMPOSITION

     FILE 'REGISTRY' ENTERED AT 15:27:41 ON 01 MAR 2003
L156           2 S 77521-29-0 OR 142008-29-5

     FILE 'HCAPLUS' ENTERED AT 15:28:25 ON 01 MAR 2003
L157          27 S L156 AND L114
L158           4 S L157 AND L121,L123,L124,L134,L153-L155
L159           9 S L155,L158
                 SEL DN AN 1 2 4
L160           3 S L159 AND E1-E9
L161          36 S L134,L155,L159 NOT L160
                 SEL DN AN 20 32
L162           2 S E10-E15
L163           5 S L160,L162 AND L112-L124,L132-L155,L157-L162
                 E EPSTEIN M/AU
L164         348 S E3-E16,E47-E50
                 E WIIG K/AU
L165           9 S E5
L166           3 S L164,L165 AND L114
                 E SENTION/PA,CS
L167           2 S E3-E6 AND L114
L168           2 S E3-E6 NOT L167
L169           8 S L166-L168,L163 AND L112-L124,L132-L155,L157-L168

     FILE 'HCAPLUS' ENTERED AT 15:35:23 ON 01 MAR 2003
                 SEL HIT RN

     FILE 'REGISTRY' ENTERED AT 15:35:45 ON 01 MAR 2003
L170          13 S E1-E13
L171           1 S 156-34-3
L172          57 S 156-34-3/CRN
L173          13 S L172 AND L18
L174          44 S L172 NOT L173

     FILE 'HCAPLUS' ENTERED AT 15:37:28 ON 01 MAR 2003

     FILE 'REGISTRY' ENTERED AT 15:37:55 ON 01 MAR 2003
L175           1 S 33817-09-3
L176          16 S 33817-09-3/CRN
L177           7 S L176 AND L18
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     FILE 'HCAPLUS' ENTERED AT 15:39:53 ON 01 MAR 2003
L178          937 S L171,L173,L175,L177
L179            5 S L178 AND L115-L120
L180           14 S L178 AND (MEMOR? OR FORGET? OR REMEMBER? OR RECALL? OR COGNIT
L181           12 S L179,L180 NOT L169
                  SEL DN AN 4 5
L182            2 S E14-E19 AND L181

     FILE 'HCAPLUS' ENTERED AT 15:43:39 ON 01 MAR 2003
L183            5 S L166,L168
L184            4 S L183 NOT PLEXUSES
                  SEL PN APPS

     FILE 'WPIX' ENTERED AT 15:44:49 ON 01 MAR 2003
L185            4 S E20-E44

     FILE 'DPCI' ENTERED AT 15:45:00 ON 01 MAR 2003
L186            0 S E20-E44

     FILE 'WPIX' ENTERED AT 15:45:10 ON 01 MAR 2003

     FILE 'WPIX' ENTERED AT 15:46:51 ON 01 MAR 2003
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